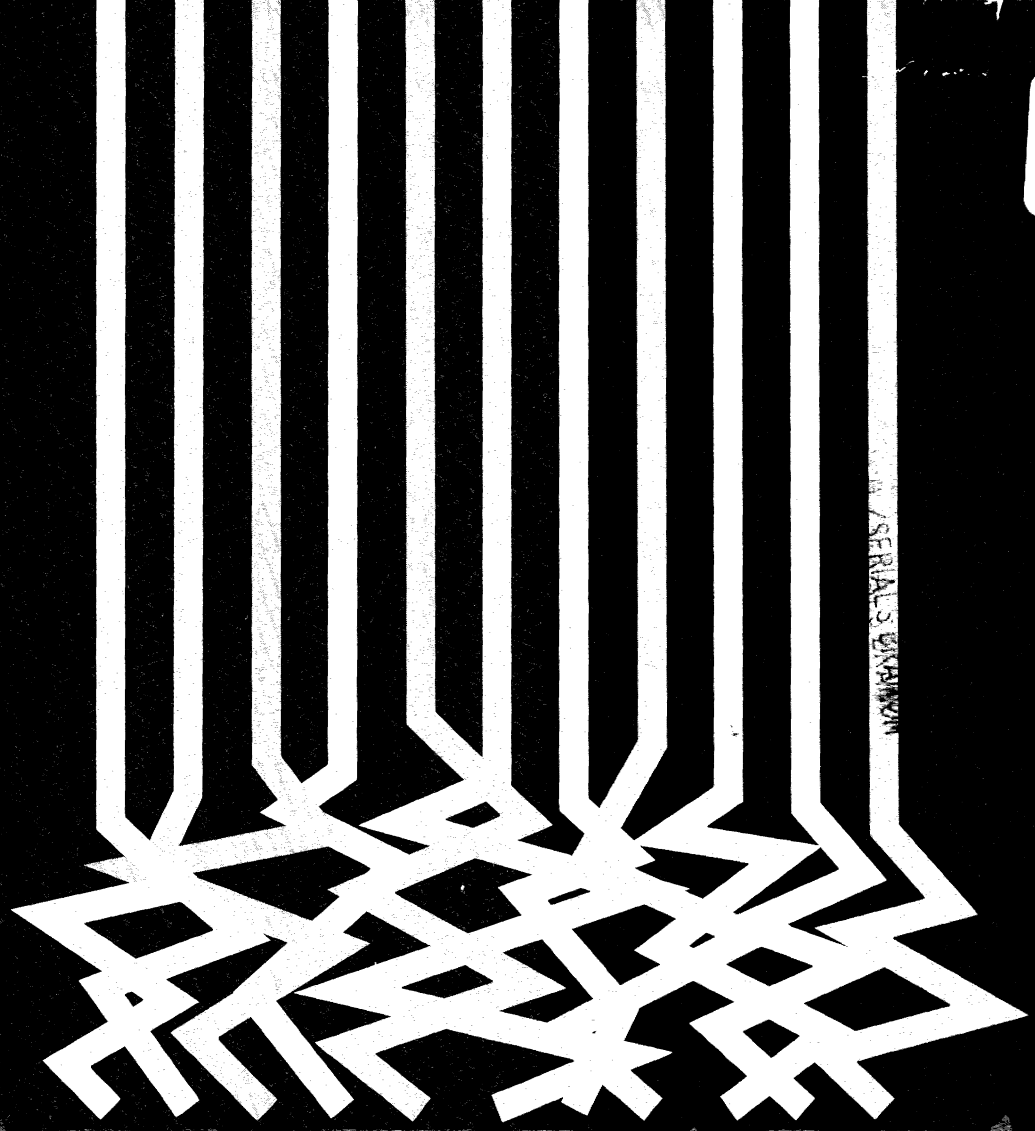

ARMILLARIA ROOT DISEASE

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ARMILLARIA ROOT DISEASE

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Preface

Armillaria root disease has been the object of intensive basic and applied study by pathologists, physiologists, taxonomists, and others since Robert Hartig published his classical work in 1874. Even with this immense collective effort, persistent confusion has obscured the real significance of *Armillaria* as a pathogen. Only recently have pathologists accepted that *Armillaria* comprises numerous species with distinct distributions and pathogenicities. This treatment resolves many contradictory claims and observations made about *Armillaria* species and the often serious root diseases they cause.

Armillaria is, however, more than just a serious pathogen. Economic importance aside, *Armillaria* possesses many fascinating biological attributes that are broached in this volume. These include bioluminescence; antibiotic and alcohol production; multiple morphological forms including rhizomorphs; in vitro fructification; peculiar mycorrhizal associations with the roots and tubers of some achlorophyllous plants; an unusual nuclear cycle; and others. In our view, the amplitude of this variability makes species of *Armillaria* well suited as experimental organisms for studying fungal development, physiology, genetics, and speciation.

Through this volume we strive to synthesize the available information on the taxonomy, physiology, and life history of *Armillaria* spp. This material is further developed to clarify the impacts, dynamics, management, and control of the root diseases caused by various species of *Armillaria* in diverse natural and exotic forests, orchards, and amenity plantings throughout the world.

The book begins with a discussion of the taxonomy and nomenclature of *Armillaria* species. Through this treatment, we not only learn how to correctly refer to these organisms but also discover why so much confusion has surrounded their taxonomy and nomenclature. This leads into chapter 2 wherein the concept and significance of biological species are explored as are the sexual patterns and life cycle of the fungus. The nutritional, biochemical, and physiological requirements of the fungus and the biochemical basis for its interactions with hosts are considered in chapter 3. Attributes of inoculum and the infection process are discussed in chapter 4. Disease symptoms and diagnosis, both on individual trees and in stands, are treated in chapter 5. Pathogenicity and various ways of assessing it are discussed in chapter 6. The next three chapters consider the role of stress factors in promoting disease and address disease development in natural forests and manmade plantations. Chapter 10 introduces mathematical modeling as a means to quantify disease development and to predict the consequences of various management actions. Chapter 11 presents management and control methods, including recent information on antagonistic organisms.

This book was conceived through discussions on *Armillaria* held among members of the International Union of Forestry Research Organizations' Working Party on Root and Butt Rots of Forest Trees. This is one of the largest, oldest, and most active IUFRO groups. Many members of that group have authored chapters for this book; many others provided ideas, advice, and encouragement. The volume stands as a tribute to the spirit of international cooperation in forestry research that is fostered by IUFRO.

The worldwide interest in, and importance of, Armillaria root disease is reflected by the contributions to this volume: 24 authors from 9 nations. Managing not only the vast amount of manuscript provided by these authors but also their often contrasting ideas, opinions, and personal reflections into a single volume with some meaningful composition and structure became our unique challenge.

Our ambition has been and remains the presentation of accurate information about *Armillaria*. Clarity of expression became the driving objective we used as a final arbiter for many difficult decisions. We wanted to remove as many potential disruptions to smooth reading as possible yet preserve an international character. Thus, we retained words and expressions unique to certain countries or cultures, but we imposed uniform spelling and punctuation standards throughout all chapters. We also sidestepped standard botanical nomenclature.

For general discussion in the text, we chose where possible to use common names as established in standard references. Coping with genus, specific epithet, authorities and multiple revisions, plus abbreviations, parentheses, and brackets proved extraordinarily tedious during manuscript preparation and revision. Ultimately, we judged the nomenclature system to be too clumsy to meet our objective of clear expression. We met the obligation for scientific accuracy by adding an Appendix which cites in alphabetical order both Latin and common names with the appropriate standard references. To overcome nomenclatural problems with reference to various *Armillaria* species, we used specific epithets only where investigators have identified their isolates. We used the generic term "*Armillaria*" where identity is uncertain.

The timing of this work seems particularly important as our knowledge of these organisms and the diseases they cause has increased markedly in recent years. We hoped that by compiling the information at this time we could stimulate and help focus further research while also providing a basis for wise and informed management of *Armillaria* diseases.

In addition to an analysis, synthesis, and consolidation of the vast literature that has accumulated, as well as the advancement of concepts and insights to assist future research on *Armillaria*, this volume celebrates the many achievements of the past. We believe this Handbook on Armillaria root disease will be of interest and value to graduate students, mycologists, pathologists, and forest managers for many years.

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Dedication

Robert Hartig (1839-1901)

The 'Father of Forest Pathology,' who concluded that wood decay was caused by microorganisms and provided convincing evidence for the pathogenicity of several fungi attacking trees. His monographic treatment of *Agaricus melleus* in Wichtige **Krankheiten der Waldbäume** (1874) has had an enduring influence on the perceptions of pathogenic behavior and study of *Armillaria*. A detailed account of Hartig's remarkable contributions to forest pathology is found in the American Phytopathological Society, English translation of this work (Phytopathological Classics No. 12, 1975).

A

C

B

D

Characteristics of Armillaria root disease. A: Infection of a seedling by rhizomorphs from an inoculum segment colonized by *Armillaria*; B: Mycelial fan in the cambial region at the base of a recently killed tree. Such fans can be diagnostic of tree death by *Armillaria*; C: *Armillaria* infection center in pole-sized ponderosa pine showing disease progression through the stand; D: Signature on an aerial photograph of an Armillaria root disease infection center. (C.G. Shaw III, R. Williams)

Contents

CHAPTER 1

Nomenclature, Taxonomy, and Identification

Roy Watling, Glen A. Kile, and Harold H. Burdsall, Jr.

Confusion has surrounded the nomenclature and taxonomy of the genus *Armillaria* (Fr.:Fr.) Staude for over a century. Until recently, taxonomists have consistently disagreed on the exact description of the genus and its correct name according to the International Code of Botanical Nomenclature. This confusion has created uncertainty for taxonomists and plant pathologists, and has hindered the study of this widely distributed and economically important genus of fungi. Based on the analyses of Watling and others (1982), we consider the genus to be a natural grouping, and that *Armillaria* is the appropriate generic name. This conclusion has been widely accepted since that publication (Antonin 1986, Bérubé and Dessureault 1988, Guillaumin and others 1985, Intini 1988, Marziano and others 1987, Rishbeth 1983, Roll-Hansen 1985, Romagnesi and Marxmuller 1983, Termorshuizen and Arnolds 1987).

The first record of an *Armillaria* species was probably either in 1729 (Micheli) or 1755 (Battarra). However, not until the later classical authors began to describe the larger fungi could several taxa now assigned to *Armillaria* in its restricted sense (*Armillaria sensu stricto*) be unequivocally recognized. From the pathologists' viewpoint, confusion has arisen from the assumption of many authors that *Armillaria mellea* (Vahl: Fr.) Kummer is a single variable or polymorphic species (Singer 1956) that occurs in both temperate and tropical regions. Although this contention is supported by maps purporting to show worldwide distribution (Distribution of Plant Diseases 143, 3rd ed. 1969) and by host lists on an international or local basis (Laemmlen and Bega 1974, Pegler and Gibson 1972, Raabe 1962a), classical European authors such as Bolton (1788-91) realized that several taxa were involved.

European interest in morphological studies of *Armillaria* was renewed in the 1970's (Romagnesi 1970, 1973, 1978; Singer 1970a,b; Singer and Clemencon 1972). The demonstration of a bifactorial sexual incompatibility system in an *Armillaria* species (Hintikka 1973) led to studies that showed several intersterile groups, termed "biological species", could be recognized in Europe (Korhonen 1978, 1980) although, as such, "biological species" had no standing within the International Code of Botanical Nomenclature. Anderson and Ullrich (1979) expanded this approach using North American isolates. Morphological and genetic data have subsequently been combined to link many biological species to morphological species and vice versa (see chapter 2). Many laboratories now consistently test interfertility to identify unknown vegetative isolates.

Armillaria probably contains about 40 species, of which several may have restricted geographical distributions or vegetation associations. The movement of phanerogams or their products from one area of the world to another may, however, have extended distributions of some species.

Species of *Armillaria* are necrotrophic pathogens of plants, and in one case of another agaric, and mycotrophic associates of achlorophyllous plants (see chapter 8). Some ecological niches recorded for members of the genus are undoubtedly exploited by several species, but the formerly broad concept of *A. mellea* applied by many authors has confounded recognition of the species involved. Retaining voucher specimens of basidiomes[1] and vegetative isolates from phytopathological studies is thus extremely important. Although the ability to identify species of *Armillaria* has advanced rapidly only in recent years, we have accumulated a wealth of observational and experimental information on various aspects of *Armillaria* biology which makes it one of our better-known genera of Agaricales.

Nomenclatural and taxonomic aspects of *Armillaria* in general and the European species in particular have been amply described in recent years (Antonin 1986,

[1]The term basidiome is used in this publication in preference to less specific terms such as basidiocarp, carpophore, fructification, fruit body, fruiting body, sporocarp, sporophore (Maas Geesteranus 1971).

Guillaumin and others 1985, Herink 1973, Marxmüller 1987, Roll-Hansen 1985, Romagnesi and Marxmüller 1983, Termorshuizen and Arnolds 1987, Watling 1987, Watling and others 1982). This chapter provides an introductory survey of the major issues in the nomenclature and taxonomy of the genus.

Armillaria (Fr.:Fr.) Staude— Nomenclature and Typification

In Fries' *Systema Mycologicum* (1821), 12 species, including *Agaricus melleus*, were accepted in the tribe *Armillaria*, which he had established 2 years earlier (Fries 1819). The tribes *Armillaria* and *Lepiota* were later combined (Fries 1825) with the latter name used for the enlarged group. However, Fries (1838) reverted to *Armillaria* for some species. By this time, the number of species in the tribe had doubled, but its scope remained unchanged in his later *Monographia Armillariarum Suecicae* (Fries 1854).

Staude (1857) was the first to raise Fries' tribe to generic rank. Singer (1951b, 1955a,b, 1986) has disputed whether Staude's entry meets all the requirements for valid publication, but Staude is now generally accepted as the validating author of the genus (Donk 1949, 1962; Watling and others 1982). Singer (1951b, 1955a,b) proposed Kummer (1871) as the correct author for *Armillaria*, and has recently reiterated that belief (Singer 1986), a conclusion we do not accept. Thus, Singer (1986) has argued that the publication of Staude (1857) is inadmissible according to the International Code of Botanical Nomenclature, but nothing has changed since Donk (1949, 1962) clearly discussed the status of Staude's account. Watling and others (1982) found no reason to disagree with Donk's findings. Both Staude and Kummer (1871) include within their generic concept *Agaricus melleus*, and as far as anyone can decide from the available information, it agrees with that outlined within Fries' (1821) tribe *Armillaria*. Fries (1821; p. 26) includes a reference to Battara (1755) under synonymy of tribe *Armillaria* but nowhere discusses this entry further. We think that this one mention can hardly support Singer's statement "defines the basic scope of the tribus." Nothing in Fries (1821) or in Battara (1755) necessitates further exploration, and this re-emphasizes the importance of *Systema Mycologicum* (Fries 1821) in forming a clear base line. Clements and Shear (1931) subsequently selected it as type species for the genus in their comprehensive survey of the nomenclature of the genera of fungi.

After accepting Staude's authority for *Armillaria*, the typification of the genus follows in a straightforward manner. Staude (1857) included four species: *Ag. mucidus*, *Ag. melleus*, *Ag. aurantius*, and *Ag. robustus*. The last two are now considered species of *Tricholoma* (Fr.) Staude, and *Ag. mucidus* is placed in *Oudemansiella* Spegazzini (or *Mucidula* Pat.). Adopting either *Ag. aurantius* or *Ag. robustus* as the type could lead to *Armillaria* becoming a synonym of *Tricholoma*. Kuhner (1988) suggested *Ag. mucidus* as the type, but this was never recommended by any earlier author. This choice would be unfortunate as *Ag. mucidus* has little in common with *Ag. melleus*. The selection of *Ag. melleus* as type by Clements and Shear (1931), Dennis and others (1954), and Donk (1949, 1962) was supported by Watling and others (1982). *Agaricus melleus* Vahl:Fr. is based on *Icones plantarum, Flora Danica* (1792), vol. 6(17): 9, plate 1013 (1790), M. Vahl (fig. 1.1) [= *Armillaria mellea* (Vahl:Fr.) Kummer in *Der Fuhrer in die Pilzkunde* (1871)]. As no herbarium specimen was available to support this plate, neotypic material was designated (Watling and others 1982).

The generic name *Armillariella* (Karsten 1881) typified by *Ag. melleus* has been used in many publications; if *Armillaria* is based on a species other than *Ag. melleus*, *Armillariella* would become available. Karsten's genus is logical if *Armillaria* is typified by *Ag. luteovirens* Alb.

FIGURE 1.1 — *Agaricus melleus*, as illustrated by Martin Vahl in Flora Danica (1790 - 1792). Marxmuller and Printz (1982) considered this figure could also represent *Armillaria borealis*, although Marxmuller (1987) accepted it as *Agaricus melleus*.

& Schw.:Fr., as supported by Singer (1951a). However, this species was not originally in Fries' tribe, a prerequisite for consideration. *Armillariella* is therefore an obligate synonym of *Armillaria*. *Floccularia* Pouzar is the correct genus for *Ag. luteovirens* and its allies.

Incorporating *Armillaria* into *Clitocybe* (Fr.) Staude has sometimes blurred the identity of what we believe to be a natural genus. While first proposed by Ricken (1915), French mycologists have most frequently followed this approach, for example Kühner and Romagnesi (1953) and Heim (1950, 1963). The latter included tropical species of *Armillaria* in his concept of *Clitocybe*. This proposal does not interfere with typification, as *Armillaria* would simply become a synonym of *Clitocybe*. However, clear differences exist in basidiome development between *Armillaria* and *Clitocybe* (Watling and others 1982). Additionally, Bennell and others (1985) showed radical differences in basidiospore wall morphology between *A. mellea* and *Clitocybe nebularis* (Batsch:Fr.) Kummer. *Clitocybe tabescens* (Scop.:Fr.) Bres. is the species usually cited as a link between the two genera. It is similar to *A. mellea* in basidiome development, basidiospore wall structure, and its bifactorial heterothallism (Anderson 1982). This species is thus best placed in *Armillaria*, probably as *A. socialis* (DC:Fr.) Herink [synonym *A. tabescens* (Scop.:Fr.) Emel.].

Singer (1951a) and Herink (1973) suggested subcategories of the genus. Singer divided *Armillaria* (as *Armillariella*) into two sections distinguished by the presence or absence of a veil (annulate and exannulate species), a subdivision he later maintained (Singer 1986). Herink (1973) followed Singer and recognized *Armillaria* as an annulate subgenus and *Desarmillaria* as an exannulate subgenus. He placed *Armillaria mellea* in the first and *A. socialis* in the second. His ideas agree with our own concepts, although we believe it will eventually be possible to subdivide the subgenus *Armillaria* into related subgroups.

Generic Characteristics

Various morphological, cultural, and other features help distinguish *Armillaria* from other genera of Agaricales. Collectively, these characters define the genus, and variations among them define species. The following are the salient characteristics of *Armillaria*:

Habit — clitocyboid with slightly sinuate, adnexed, subdecurrent or decurrent gills; bivelangiocarpic or metavelangiocarpic development in annulate species, apparently monovelangiocarpic development in exannulate species; solitary, gregarious, or caespitose.
Pileus — fleshy, thinning towards margin, expallant, hygrophanous or not; color variable yellow-brown, yellow-olivaceous, ochraceous, rusty-tawny, umber, cigar brown, less commonly buff or clay pink, sometimes ivory, pallid, or even mouse-gray; surface glabrous, scurfy, squamulose, squamules darker than ground color, sometimes restricted to disc; glabrescent as scales are lost; dry or becoming viscid to distinctly viscid, in some species almost glutinous.
Stipe — central, fibrous-fleshy, not characteristically cartilaginous; often becoming hollow and the outermost layers splitting and curling back to expose flesh; more or less annulate with floccose-membranous to arachnoid veil; often arising from sheets of white mycelia or from well-differentiated black rhizomorphs, and/or, associated with plaques of thin, black, tough tissue.
Lamellae — close to subdistant; moderately thick; nearly white, ivory, or cream-color at first but frequently becoming spotted with cinnamon-buff, rusty-tawny, or sometimes, particularly with age, with a tinge of purple or distinctly pink; sinuate; adnexed to deeply decurrent.
Flesh — of pileus pale and of stipe white at first, becoming as dark as umber or Vandyke brown downwards and sometimes tinted red or bluish at base where colonized by pigment-producing bacteria or nectriaceous fungi.
Spore-print — white to cream-color darkening slightly on drying, and in herbarium material.
Basidia — 4-spored, sometimes 2-spored; thin-walled; with or without a basal clamp-connection; hyaline; smooth-walled in aqueous alkali solutions or if thick-walled [= crassobasidia (Chandra and Watling 1983)] then appearing silvery or glassy, and/or, becoming ochraceous or fulvous.
Basidiospores — ellipsoid; inamyloid; hyaline, yellowish cream-color or ochraceous in aqueous alkali solutions; weakly cyanophilic; thin to moderately thick-walled; smooth or slightly verruculose or rugulose with broad, blunt usually prominent apiculus; lacking germ-pore or apical differentiation (thinning or thickening).
Cheilocystidia — present or absent, often inconspicuous; variable in shape sometimes catenulate-septate; thin-walled or becoming slightly thick-walled with age sometimes with apical prolongation and with or without basal clamp-connection; smooth; hyaline to honey-colored in aqueous alkali solutions.
Pleurocystidia — absent or, if present, thin-walled; poorly differentiated and rarely visible above the level of the basidia.
Pileipellis — an irregular, disrupted trichodermium consisting of (i) an irregular, easily destroyed *suprapellis* composed of groups of fulvous or cinnamon, subparallel, ascendant, loosely to strongly

adhering hyphae intermixed with broad, frequently encrusted hyphae (which form the scales), often with clamp-connections; ascendant hyphae becoming repent to form a rather amorphous adnate layer; (ii) *mediopellis* - of parallel to subparallel hyphae forming a cutis that may or may not gelatinize but sooner or later becomes the outermost layer; and (iii) *subpellis* - a compact hyphal layer.

Stipitipellis — parallel hyphae overlain by more or less strongly developed, irregular, filamentous velar remnants; in parts of stipe free from velar material showing development of cylindric to elongate clavate or lageniform caulocystidia.

Pileus and stipe trama — monomitic; hyphae inamyloid, generally lacking clamp connections.

Hymenophoral trama — bilateral at first and remaining so or becoming regular with age although always demonstrating some divergent arrangement; constitutive hyphae generally lacking clamp-connections; inamyloid.

Vegetative growth — variable on agar media but typically reddish-brown crustose surface mycelium; usually slow growing; with or without tufts of cinnamon aerial mycelium; with or without reddish-brown rhizomorphs or with white to cream-color rhizomorphs embedded in the medium with emergent reddish-brown tips; rhizomorphs branch monopodially, dichotomously, or irregularly; vegetative mycelium often bioluminescent; cells uni- or multinucleate; nuclei apparently diploid.

Rhizomorphs — mycelial aggregations with a melanized outer layer and pale, apical growing tip; produced in culture and from infected lignicolous material.

Single basidiospore isolates — from heterothallic species typically slow growing; producing white, fluffy to cottony mycelium, sometimes with areas of brown or reddish; with or without sparse rhizomorph development; nuclei haploid.

Compatibility system — bifactorial; heterothallic with multiple alleles at the incompatibility loci; some species possibly homothallic.

Relationships With Other Agarics

Modern classifications of the Agaricales link *Armillaria* s.s. with the Tricholomataceae (Jülich 1981; Kühner 1980; Singer 1951a, 1986). However, even in the temperate northern hemisphere where the agarics have been most intensively studied, only Jülich (1981) indicated a strong relationship between *Armillaria* and another genus in the Tricholomataceae, *Tricholomopsis* Singer. Possible relationships to the Cystodermataceae (Romagnesi 1980), the Entolomateaceae (Bennell and others 1985), and the Amanitaceae (Helfer and Watling 1989) also have been discussed.

The many distinctive morphological characteristics of the genus, the production of characteristic rhizomorphs, both parasitic and saprophytic capabilities, and the apparently diploid nuclei in the vegetative mycelium (see chapter 2), lead us to believe that it stands quite distantly from other agaricoid genera. Thus, Jülich's (1981) introduction of the family Armillariaceae to accommodate the genus has great merit.

Relationships Within *Armillaria*

Apart from the subgeneric distinction between developmental patterns in annulate and exannulate species and its inference of relatedness, no systematic attempt has been made to assess the phylogeny of species based on differences in morphology, physiology, biochemistry, ecology, pathology, or sexual compatibility system. Computer-aided comparative studies of such attributes could assist research into species relatedness.

Divergent nucleic acid composition has probable utility in ascertaining species relatedness. Anderson and others (1987) concluded that some particular DNA sequences may be appropriately variable for phylogenetic studies. Subsequently, Anderson and others (1989) showed that some European *Armillaria* species and the equivalent or unidentified North American Biological Species, or NABS, (Anderson and Ullrich 1979; Bérubé and Dessureault 1988, 1989) could be placed in distinct classes based on restriction maps of ribosomal DNA. These are: rDNA class 1, *A. ostoyae* (= NABS I); class 2, *A. gemina* (= NABS II); class 3, *A. borealis*; class 4, *A. sinapina* (= NABS V); NABS IX, X; class 5, *A. calvescens* (= NABS III), *A. gallica* (= NABS VII), *A. cepistipes* (= NABS XI?); class 6, *A. mellea* (= NABS VI). The classes are believed, with the possible exception of rDNA class 4, to represent natural groupings. In addition, classes 1, 2, and 3 were considered to be closely related with rDNA classes 2 and 3 derived from the more widely distributed DNA class 1. Greater resolution through detailed mapping of particular regions of the genome will assist phylogeny development. As Anderson and others (1989) have suggested, reconsidering ecological, morphological, and distributional data for taxa on the basis of restriction polymorphisms would be informative.

Present and Excluded Species of *Armillaria*

Singer (1978) prepared a key to the world taxa (as *Armillariella*) he considered distinct. This key needs to be updated in light of the new taxa recognized and concepts developed since that time. Table 1.1 lists 36 taxa which we believe have been documented suffi-

TABLE 1.1 — The current nomenclature and geographical occurrence of 36 *Armillaria* species (some as *Armillariella*). The citation for the original description of each species is given. Italic numbers indicate those identified as both morphological and biological species.

1. *A. mellea* (Vahl:Fr.) Kummer (= Korhonen D., Anderson and Ullrich NABS VI). Europe, North America, North Asia, Japan, Africa? (type species)+.
2. *Armillariella affinis* Singer. Central America. In Fieldiana (Bot.).21:12 (1989).
3. *A. borealis* Marxmüller & Korhonen (= Korhonen A.). Northern Europe, Russia. In Bull. Soc. Mycol. Fr. 98:122 (1982).
4. *A. calvescens* Bérubé & Dessureault (= Anderson and Ullrich NABS III). North America. In Mycologia. 81:220 (1989).
5. *A. cepistipes* Velenovsky (= Korhonen B., Anderson and Ullrich (Morrison) NABS XI?). Europe, North America?, Japan. In Ceske Houby. 1:283 (1920).
6. *A. fellea* (Hongo) Kile & Watling. New Guinea. In Rep. Tottori Mycol. Inst. 14:97 (1976).
7. *A. fuscipes* Petch (= *A. heimii* Pegler and *A. elegans* Heim). East and West Africa, Sri Lanka, Madagascar. In Ann. Roy. Bot. Gdn., Peradeniya. 4:299 (1909). †
8. *A. gallica* Marxmüller & Romagnesi (= *A. lutea* Gillet sensu Arnolds and Temorshuizen, and Watling; *A. bulbosa* (Barla) Kile and Watling; Korhonen E., Anderson and Ullrich NABS VII). Europe, North America, Japan. In Bull. Soc. Mycol. Fr. 103:152 (1987).#
9. *A. gemina* Bérubé & Dessureault (= Anderson and Ullrich NABS II). North America. In Mycologia. 81:217 (1989).
10. *Armillariella griseomellea* Singer. South America. In Beih. Nova Hedw. 29:40 (1969).
11. *A. hinnulea* Kile & Watling. South-eastern Australia. In Trans. Brit. Mycol. Soc. 81:131 (1983).
12. *A. limonea* (Stevenson) Boesewinkel. New Zealand. In Kew. Bull. 19:13 (1964).
13. *A. luteobubalina* Watling & Kile. Australia. In Trans. Brit. Mycol. Soc. 71:79 (1978).
14. *A. mellea* var. *camurenensis* Henning. West Africa. In Engl. Bot. Jahrb. 22:107 (1895).
15. *A. melleorubens* (Berkeley & Curtis) Saccardo. Caribbean. In J. Linn. Soc. 10:283 (1869).
16. *A. macrospora* Peck. North America. In Bull. Torrey Bot. Club. 27: 610 (1900).
17. *A. montagnei* (Singer) Herink. South America. In Lloydia. 19:182 (1956).
18. *A. nigritula* Orton. Great Britain. In Notes Roy. Bot. Gdn., Edinb. 38:316 (1980).
19. *A. novae-zelandiae* (Stevenson) Herink. New Zealand, Eastern Australia, New Guinea, South America? In Kew Bull. 19:14 (1964).
20. *A. olivacea* (Rick.) Herink. South America. In Lloydia. 19:180 (1956).
21. *A. omnituens* (Berkeley) Saccardo. India. In Hooker's J. Bot. 2:46 (1850).
22. *A. ostoyae* (Romagnesi) Herink (= *A. polymyces* (Secr.) Sing. & Clem; *A. obscura* Schaeff.:Fr., *A. montagnei* var *umbrinolutea* Singer, = Korhonen C; Anderson and Ullrich NABS I). Europe, North America, Japan. In Bull. Soc. Mycol. Fr. 86:265 (1970).
23. *A. pallidula* Kile & Watling. Queensland. In Trans. Brit. Mycol. Soc. 91:307 (1988).
24. *A. praecox* Velenovsky. Central Europe. In Ceske Houby. 1:282 (1920).
25. *A. procera* Speggazini. South America. In Bol. Acad. Nac. Cienc.Cordoba. 11:385 (1889).
26. *A. puiggarii* Speggazini. South America. In Bol. Acad. Nac. Cienc. Cordoba. 11:384 (1889).
27. *A. saviczii* (Singer) Herink. Byelorussia. In Nat. Syst. Sect. Crypt. Inst. Bot. Acad. Sci. URSS. 4(10-12):6 (1938).
28. *A. sinapina* Bérubé & Dessureault (= Anderson and Ullrich NABS V). North America. In Can. J. Bot. 66:2030 (1988).**
29. *A. solidipes* Peck. North America. In Bull. Torrey Bot. Club. 27:611 (1900).
30. *A. sparrei* Singer (Herink). South America. In Lloydia. 19:183 (1956).
31. *Armillariella tigrensis* (Singer) Raith. South America. In Flora Neotropica Monogr. 3:8 (1970).
32. *A. yungensis* (Singer) Herink. South America. In Flora Neotropica Monogr. 3:12 (1970). Subgenus Desarmillaria
33. *A. ectypa* (Fr.) Lamoure. Europe. In Syst. Mycol. I:108 (1821).
34. *A. nigropunctata* (Secretan) Herink. Europe. In Mycogr. Suisse. 2: 1046 (1833).
35. *A. socialis* (DC.:Fr.) Herink. (= *A. tabescens* (Scop.:Fr.) Emel.). Europe, USA? In Syst. Mycol. I:251 (1821).*
36. *Armillariella watsonii* (Murrill) Singer. North America. In Proc. FL. Acad. Sci. 7:111 (1944)

\+ For species 1,3,4,5,8,9,22, and 28, the secondary designations given are those used for the equivalent biological species by Korhonen (1978), Anderson and Ullrich (1979), and Morrison and others (1985).

† Synonymy proposed by Kile and Watling (1988) on morphological criteria, although interfertility studies are required for confirmation.

** *A. sinapina* (NABS V) may be synonymous with *A. cepistipes* (Anderson and others 1980, Guillaumin and others 1989a) but comparisons of basidiome morphology and further interfertility studies between European and North American material are necessary to resolve this question.

\# The binomial *A. gallica* is preferred as its identity is unequivocal, being supported by a type specimen, a culture, a full description, and a plate.

* While the name *A. (Clitocybe) tabescens* has been frequently used for a taxon common as a pathogen in southeastern USA, it is probably a different species than that found in Europe (Guillaumin and others 1989a).

ciently to be considered species, although a few additional taxa will probably be delineated eventually. It includes all species known to be significant to plant pathologists and ecologists. Nomenclatural adjustment of some of Singer's *Armillariella* species is required. Fourteen of the species have been recognized as both morphological and biological species (see chapter 2), and future interfertility-morphological studies may result in changes to the status of other species listed in table 1.1.

Since Fries (1821), many species have been placed in *Armillaria* by virtue of possessing a white to cream-color spore-print and an annulus, which make it very heterogenous. With a more restricted generic concept for *Armillaria*, knowing where some of these taxa formerly placed within *Armillaria* are now assigned is useful. Table 1.2 shows the concordance of Fries (1821) species with modern concepts. Fries (1838, 1854, 1874) included an additional 34 species in the *Armillaria* group, only one of which was possibly an *Armillaria* species s.s. (*A. laricinus* = *A. ostoyae?*). Many velate species of *Tricholoma* have been placed in *Armillaria*, and *T. caligata* (Viv.) Rick. and its allies have been traditionally placed by North Americans in the genus (Hotson 1941, Mitchel and Smith 1976, Smith 1979, Thiers and Sundberg 1976). This is erroneous and confusing because the species are morphologically, ecologically, and biologically quite distinct from *Armillaria* species s.s.

Romagnesi (1970, 1973), Termorshuizen and Arnolds (1987), Watling (1987), and Watling and others (1982) discussed the identity of *Armillaria* species illustrated in the classical literature.

TABLE 1.2 — Concordance of Fries' Systema Mycologicum (1821) species in Agaricus Tribe III *Armillaria* with modern concepts.

Species	Family
1. *A. robustus* = *Tricholoma*	Tricholomataceae
2. *A. persoonii**	
3. *A. guttatus* = *Limacella*	Amanitaceae
4. *A. bulbiger* = *Leucocortinarius*	Cortinariaceae
5. *A. constrictus* = *Calocybe*	Tricholomataceae
6. *A. subcavus* = *Limacella*	Amanitaceae
7. *A. mucidus* = *Oudemansiella*	Tricholomataceae
8. *A. vagans**	
9. *A. griseofuscus**	
10. *A. denigratus* = *Agrocybe erebia*	Bolbitiaceae
11. *A. rhagodiosus* = *Lentinus lepideus*	Pleurotaceae
12. *A. melleus*	

* *A. persoonii*, *A. vagans*, and *A. griseofuscus* cannot be equated with any modern taxa and are best considered *nomen dubium*.

Recent major contributions to the description of morphological variation and the delineation of *Armillaria* taxa include those of Romagnesi (1970, 1973, 1978); Marxmüller (1982, 1987); Marxmüller and Printz (1982); Romagnesi and Marxmüller (1983); Watling (1987) for Europe; Singer (1956, 1969) for South America; Stevenson (1964) and Kile and Watling (1981, 1983, 1988) for Australasia; and Bérubé and Dessureault (1988, 1989) for North America. Although Chandra and Watling (1982) redescribed several Indian species, fresh collections are required to complement their herbarium studies. Mohammed and others (1989) and Mwangi and others (1989) reported cultural, genetic, and isozyme studies of African species which will help to resolve their identity. Further research is necessary for other areas such as Siberia, China, and parts of Southeast Asia.

Taxonomic Characters and Identification

As with other macromycetes, species of *Armillaria* are delimited primarily by basidiome morphology (fig. 1.2). While vegetative isolates may be identified or grouped by various methods, basidiomes are essential for the complete description and naming of species.

Basidiome macromorphology, pileipellis structure and ornamentation, ring characteristics, stipe ornamentation, presence or absence of subhymenial or basidial clamps, location of pigments in cell walls or vacuoles, and basidiospore size and ornamentation are among characters of value for species differentiation. Separation of some species by morphological criteria alone is difficult but no more so than in many other agaric genera. Identification may require using numerous macro- and micromorphological features combined with biochemical, cultural, and ecological information. A thorough appreciation of the most useful taxonomic characters will only be derived from careful analyses of all these features (Watling and others 1982).

Analysis of European, and to a lesser extent Australasian species (Kile and Watling 1983, Shaw and others 1981), showed that it is possible to identify some species by morphological and physiological attributes of their vegetative mycelia and rhizomorphs as well as by basidiome morphology (table 1.3). Additional simple tests such as the response of the mycelium to light may also differentiate some species (Benjamin 1983; see also Hood and Sandberg 1987).

Serological differences among several *Armillaria* species were demonstrated by Lung-Escarmant and others (1978, 1985b) and Lung-Escarmant and Dunez (1979, 1980); serological techniques may, in the future, have a substantial impact on the delimitation of *Armillaria*

FIGURE 1.2 — Basidiomes of 12 *Armillaria* species from various regions of the world, demonstrating variation in the macromorphology of basidiomes.
A: *A. ostoyae*; B: *A. limonea*; C: *A. novae-zelandiae*; D: *A. pallidula*; E: *A. mellea*; F: *A. fumosa*; G: *A. calvescens*; H: *A. luteobubalina*; I: *A. gallica*; J: *A. sinapina*; K: *A. tabescens*; L: *A. ostoyae* produced in vitro. (G.A. Kile, H. Burdsall, A. Lynch, J. Worrall, P. Wargo, C.G. Shaw III, T. Harrington)

TABLE 1.3 — Morphological, physiological, and serological differences among *Armillaria* species common in Europe (*A. mellea, A. borealis, A. cepistipes, A. gallica, A. socialis,* and *A. ostoyae*).*

	Differences between species	References
1. Morphology of basidiomes in nature	All species different Difficult distinction between *A. gallica* and *A. cepistipes*	Romagnesi 1970, 1973 Marxmüller 1982, 1987 Romagnesi and Marxmüller 1983 Roll-Hansen 1985 Motta and Korhonen 1986 Watling 1987 Intini 1988
2. Morphology of basidiomes in vitro	Useful for *A. ostoyae*, *A. borealis* and *A. cepistipes*	Guillaumin 1986a
3. Morphology of the mycelium in pure culture	All species different except *A. gallica* and *A. cepistipes*	Korhonen 1978 Guillaumin and Berthelay 1981 Rishbeth 1986 Mohammed 1987 Intini and Gabucci 1987 Guillaumin and others 1989a
4. Morphology of subterranean rhizomorphs in nature	*A. ostoyae, A. mellea* and *A. gallica* different	Morrison 1982
5. Morphology of subterranean rhizomorphs in a mist box	All species different except *A. gallica* and *A. cepistipes*	Mohammed 1985, 1987 Guillaumin and others 1989a
6. Response to temperature	Different temperature optima. Poor growth of *A. mellea* but good growth of *A. socialis* at 30 degrees C	Rishbeth 1986 Mohammed 1987
7. Reaction to phenolic acids and terpenes	Specific reaction of *A. gallica*, others quite variable	Shaw 1985 Rishbeth 1986 Mohammed 1987 Guillaumin and others 1989a
8. Polyclonal antibodies	Separate *A. mellea*, *A. gallica, A. ostoyae, A. socialis*	Lung-Escarmant and Dunez 1979, 1980 Lung-Escarmant and others 1978,1985

*Modified from Guillaumin

species. Fox and Hahne (1989) used monoclonal antibodies, but the results to date are not as impressive as those obtained by studies using polyclonal antibodies. Refinement of the techniques by developing greater antibody specificity to overcome problems of cross reactivity between closely related species may allow accurate identification in the near future, including the possibility of diagnostic kits for rapidly identifying field material.

Nucleic acid analysis supports current species concepts in *Armillaria*, and offers a powerful diagnostic tool. Motta and others (1986) reported quantitative differences in nuclear DNA content between *A. mellea* and *A. gallica*. Jahnke and others (1987) and Anderson and others (1987, 1989) showed that mitochondrial (mt) DNA was highly conserved within species but divergent between them, and that restriction fragment patterns were therefore diagnostic for species. Smith and Anderson (1989) correctly identified 23 North American isolates using DNA restriction fragment length polymorphisms.

Isoenzyme and protein profiles of some northern hemisphere taxa also differ sufficiently to offer further methods of species separation (Lin and others 1989, Lung-Escarmant and others 1985b, Morrison and others 1984).

The biological species concept has been applied to the genus using single basidiospore isolates to delineate reproductively isolated groups as discussed in chapter 2. Using this particular approach has greatly assisted taxonomists in defining species in genera with restricted interspecific but high intraspecific morphological variation. Reproductively isolated groups have been linked to existing taxa (Marxmüller 1982, Romagnesi and Marxmüller 1983), led to the description of new taxa (Bérubé and Dessureault 1988, 1989; Marxmüller and Korhonen in Marxmüller 1982; Marxmüller 1987), and established intra- (Anderson and Ullrich 1979, Kile and others 1983, Korhonen 1978) and inter-continental distributions (Anderson and others 1980, Guillaumin and others 1989a, Morrison and others 1985a). Conversely, species initially described on conventional criteria were later shown to be biological species (Guillaumin 1986a, Kile and Watling 1988).

Cumulative experience suggests that reconciling morphological (taxonomic) and biological species concepts for most *Armillariae* will be possible. Although such studies will take time to complete, they should result in robust characterization of species. In cases for which detailed morphological examination supports a single species but interfertility studies indicate otherwise, Watling and others (1982) suggested adopting the macro-microspecies concept in which a macrospecies would consist of morphologically indistinguishable biological species. We support this suggestion.

Conclusions

Major studies of *Armillaria* taxonomy have been completed in recent years. Linking morphological, cultural, physiological, and genetic data has often enhanced their individual values; the frequent concordance of information from a variety of sources has more clearly defined many taxa. Additional collections and application of various techniques to assess phenotypic and genotypic variation within the *Armillaria* flora in regions where it is incompletely known remain necessary to enhance our taxonomic understanding of the genus on a worldwide basis. Analysis of collections on which some early names are based will further assist the quest for nomenclatural stability within the genus.

The genetic approach to species differentiation, initiated for *Armillaria* by Korhonen (1978), allowed the identification of species from vegetative isolates. Subsequent work has shown that vegetative isolates also may be distinguished by other cultural or physiological characteristics. The ability to identify vegetative isolates is highly useful for organisms in which the vegetative phase may often be the only one encountered. Newer techniques such as DNA analysis and production of monoclonal antibodies have the potential to further enhance rapid and reliable identification of vegetative isolates.

The morphological and biological species concepts appear largely reconcilable for *Armillaria*, at least on the basis of our knowledge of temperate species. This perhaps fortuitous situation will continue to have a marked impact in clarifying the taxonomy of the genus.

A stable nomenclature, well-defined species, and a variety of identification techniques are invaluable to pathologists and ecologists in their attempts to understand the behavior and natural relationships of *Armillaria* species, clarify their natural relationships, and develop disease-control strategies. Progress has been significant in the former areas in recent years.

CHAPTER 2

Life Cycle, Interfertility, and Biological Species

Jean-Jacques Guillaumin, James B. Anderson, and Kari Korhonen

Species are traditionally identified by their morphological characteristics. Within the last few decades, however, the "biological species" concept has assumed an increasingly important role in mycology. A biological species is a group of "individuals" sharing a common gene pool. In the field, there is little or no genetic exchange between biological species (Esser and Hoffman 1977). Although the biological species is a rather limited concept dependent only on the criterion of interbreeding, it has already had a major impact on formal taxonomy. Among basidiomycetes especially, interfertility tests very often conclusively indicate species identity (Boidin 1977, Boidin and Lanquetin 1984). Of course, interfertility tests can only be conducted with sufficient knowledge of the sexual incompatibility systems and life cycles of the fungal group under investigation. In the genus *Armillaria*, interfertility tests became possible only when the riddle of sexuality was solved, beginning with Hintikka in 1973.

In the Basidiomycetes, single basidiospores germinate to produce a mycelium usually consisting of haploid, monokaryotic (uninucleate) cells. In heterothallic species, haploid monokaryons anastomose with one another upon contact; if they are sexually compatible, a fertile mycelium usually consisting of dikaryotic (binucleate) cells results. In many, but not all, species, the synchronous division of the paired nuclei in a dikaryon accompanies the formation of clamp connections, the presence or absence of which is the most widely used criterion for judging whether a pairing of haploid monokaryons is sexually compatible or incompatible.

The dikaryon predominates in the vegetative phase of most basidiomycetes. During vegetative growth, the two component nuclei remain paired but do not fuse. Only in the basidia does nuclear fusion (karyogamy) finally occur immediately before meiosis and the formation of basidiospores (fig. 2.1).

Most basidiomycetes are heterothallic. The haploid monokaryon is self-sterile, and a dikaryon appears only when two haploid monokaryons carrying different alleles at the mating-type locus or loci contact one another and mate. "Unifactorial" species have one mating-type locus, and the monospore isolates from a single basidiome segregate as two classes or "mating types" ("bipolar pattern of sexuality"). "Bifactorial" species have two mating-type loci, and the monospore isolates from a basidiome segregate as four mating-types ("tetrapolar pattern of sexuality").

A few basidiomycetes are homothallic. The haploid monokaryon is self-fertile, and becomes dikaryotic and fertile even without mating with another strain. "Pseudohomothallic" species have a uni- or bifactorial sexual incompatibility system, but individual basidiospores may receive two postmeiotic nuclei carrying

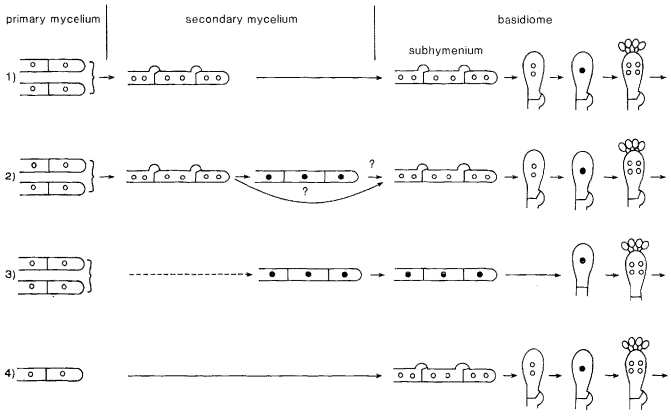


FIGURE 2.1 — Caryological cycles: 1) a typical hymenomycete with dikaryotic secondary stage; 2) a heterothallic *Armillaria* with dikaryotic subhymenium; 3) a heterothallic *Armillaria* with diploid subhymenium; 4) a homothallic *Armillaria* (*A. ectypa*; there are also homothallic *Armillaria* species with monokaryotic subhymenium). Open circles are haploid nuclei, dark circles diploid. The cycles of *Armillaria* are somewhat hypothetical.

compatible mating types. The resulting monospore isolates of these species are self-fertile but for a different reason than in true homothallic species.

Some early researchers (Kniep 1911, Kühner 1946) observed that *Armillaria* did not fit the general concept of the higher basidiomycete life cycle. They noted the hyphal cells of *Armillaria* are monokaryotic, irrespective of whether the culture originates from a single basidiospore, basidiome tissue, or vegetative material from the field. One plausible explanation was that *Armillaria* is homothallic. An observation inconsistent with homothallism and inbreeding, however, was that monospore mycelia originating from a single basidiome vary considerably, suggesting meiosis and recombination in a heterozygous parent (Raabe 1953, Snider 1957). The state of knowledge of the *Armillaria* life cycle was aptly summarized by Raper (1966): "All criteria point to an asexual or homothallic pattern of development, save one: the variability among the monosporous progeny of single fruiting bodies."

The Sexual System

Mating Reactions Among Haploids

Hintikka (1973) made the first and most important contribution to solving the problem of sexual reproduction in *Armillaria*. He observed a macromorphological difference between monospore and tissue cultures of *Armillaria*. The monospore isolates usually produce a white or light-brown aerial mycelium which gives the colony a fluffy appearance. Cultures from basidiome tissues, however, are flat, crustose, and lack aerial mycelia. Based on this morphological distinction, Hintikka showed that *Armillaria* had a bifactorial sexual incompatibility system. When sibling monospore isolates were confronted in culture, the colony morphology of certain pairwise combinations changed from the fluffy to the flat and crustose appearance. Also, because the cells both of unmated monospore isolates and of basidiome tissues are monokaryotic, he suspected that the nuclei in crustose mycelia were diploid. Diploidization in matings was proved later by several different lines of investigation.

According to the bifactorial sexual incompatibility system, each haploid mycelium of *Armillaria* contains two mating-type alleles, *A*x and *B*x. After two haploid mycelia (belonging to the same species) contact one another and anastomose, one of four possible events may occur (fig. 2.2):

(1) **Incompatible mating** [A_1B_1xA_1B_1]: The haploid partners grow side by side without intermingling, and without any substantial changes in macro- or micromorphology.
(2) **Compatible mating** [A_1B_1xA_2B_2]: The partners intermingle eventually to form a homogeneous colony while the morphology changes from the fluffy to the flat, crustose type. Partially disintegrated septa are visible in some hyphae, indicating nuclear migration. Most species also have some dikaryotic hyphae with clamp connections. Nuclear migration and diploidization proceed rather slowly in matings of *Armillaria*, only about 2-3 times faster than the growth of hyphae (Korhonen 1983).
(3) and (4) **Hemicompatible common-*A* and common-*B* matings** [A_1B_1xA_1B_2 and A_1B_1xA_2B_1]: One of these combinations is similar to an incompatible mating, but in the other combination, a broad "barrage" zone usually develops between the partners. Aerial mycelium is sparse or lacking, and sometimes the crustose mycelial type is also seen in this zone. Some ambiguity persists about the assignment of *A* and *B* factors, however. According to one interpretation, the latter hemicompatible interaction is common-*A* because signs of nuclear migration (disintegrated septa) can be found in some hyphae of the barrage zone, suggesting the presence of different-*B* alleles (Korhonen 1978). According to the other interpretation, the crustose mycelium on the barrage zone is a common-*B* diploid (Guillaumin and others 1983).

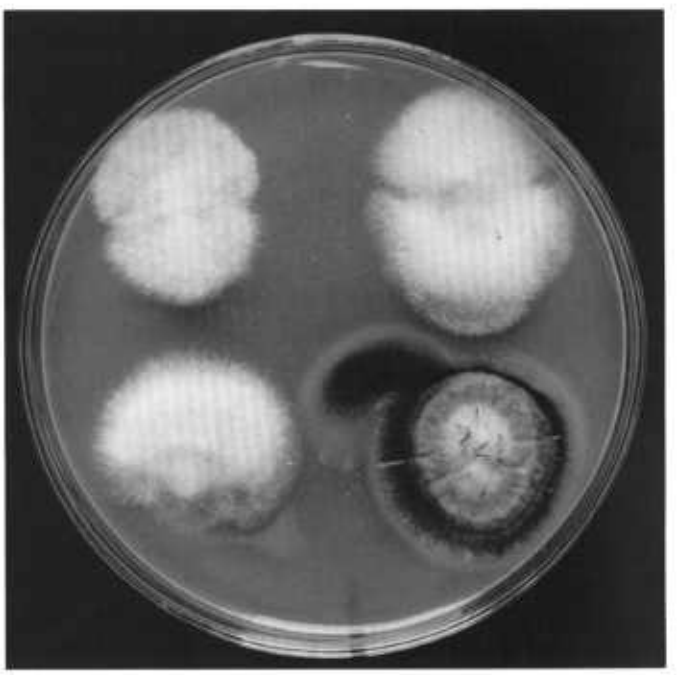


FIGURE 2.2 — Appearance of different incompatibility factor combinations in matings of *A. ostoyae*: incompatible, two hemicompatible, and compatible matings (from upper left to lower right). Age of cultures: 6 weeks. (J. Anderson)

When single-spore isolates from one basidiome are paired with each other, these four mating factor combinations appear at about equal frequencies. The great majority of pairings within a large population are compatible because the number of different alleles in the population is large, and because in any given pairing of nonsiblings collected from different localities their alleles are unlikely to be identical. No reliable estimates gauge the total number of different mating-factor alleles in the species of *Armillaria*. As judged on the basis of some large mating tests, the number must be several dozen at least. In this respect, *Armillaria* is similar to other bifactorially heterothallic basidiomycetes.

The existence of the same bifactorial sexual incompatibility system has now been shown in all temperate *Armillaria* species (Guillaumin 1986a, Guillaumin and others 1983, Kile 1983b, Kile and Watling 1988, Korhonen 1978, Ullrich and Anderson 1978) that have been investigated, except for the very rare Eurasian species *Armillaria ectypa* (Korhonen unpubl., Guillaumin unpubl.).

Matings Between Diploids and Haploids

A process analogous to the Buller phenomenon exists in *Armillaria* (Anderson and Ullrich 1982a; Korhonen 1978, 1983). When a fluffy haploid mycelium is paired with a crustose diploid isolate of the same species, in many cases the morphology of the former progressively changes to crustose, indicating diploidization. The Buller phenomenon in its original sense (Raper 1966) is a mating between a monokaryon and a dikaryon: the dikaryon donates compatible haploid nuclei to the monokaryon, which is "dikaryotized." In *Armillaria*, the donor mycelium is diploid; the exact mechanisms of diploid-haploid mating are not known. In most cases, the diploid nuclei apparently replace the haploid nuclei in the opposing mycelium; occasionally, however, recombinant diploids appear, indicating that haploidization has taken place in the original diploids (Guillaumin 1986a).

The Caryological Cycle

Vegetative Diploidy

In a typical basidiomycete, the final result of compatible mating is a heterokaryotic mycelium with two or more haploid nuclei in each cell. In the genus *Armillaria*, the result is a diploid mycelium with uninucleate cells although the cells in older parts of the mycelium, in rhizomorphs, and in basidiomes, are commonly multinucleate.

When two haploid, monokaryotic cells mate, they first unite to form a dikaryotic stage with binucleate cells and clamp connections (fig. 2.1). This stage is only transient in *Armillaria*. Within a few days, the isolated dikaryotic hyphae become monokaryotic. This change is caused by somatic nuclear fusion and diploidization in the tip cells. After nuclear fusion, the cell undergoes mitotic division. This peculiar cell division produces two monokaryotic diploid cells from one dikaryotic cell (fig. 2.3). The diploid tip of the hypha continues to grow and dikaryotic cells are no longer apparent (Anderson 1982, Korhonen 1983, Korhonen and Hintikka 1974). Despite the instability of the dikaryotic hyphae, they can be cultivated by transferring dikaryotic tips repeatedly to a new medium (Korhonen and Hintikka 1974).

This mating process has been observed in several species of *Armillaria* including *A. borealis*, *A. gallica*, *A. cepistipes*, *A. ostoyae*, and *A. tabescens*. All of these species have a transient, but distinct, dikaryotic stage in compatible matings (Anderson 1982, Guillaumin 1986a, Korhonen 1978). The mating process in *A. mellea* seems to be somewhat different. A dikaryotic stage has never been found (figs. 2.1-2.3), and the diploidization mechanism in this species is unclear (Guillaumin 1986a).

Several additional lines of evidence show that the vegetative stage of *Armillaria* is diploid. In *A. ostoyae*, auxotrophic mutants with various nutritional deficiencies have been recovered from haploid, single-spore isolates and used as markers to investigate the mating

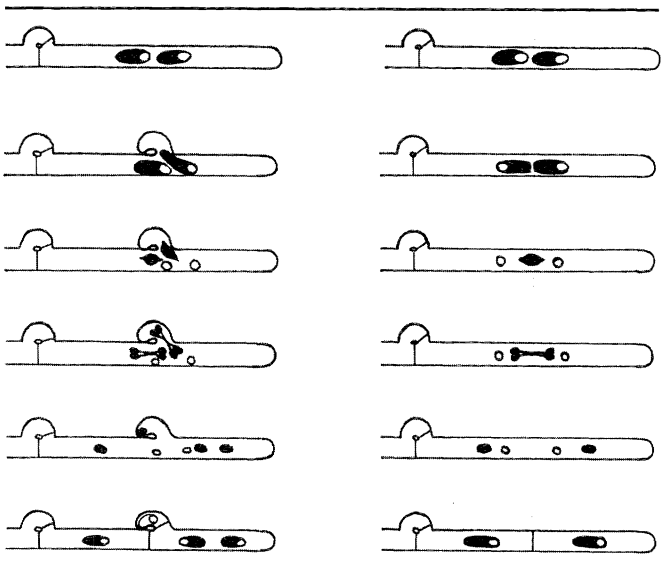


FIGURE 2.3 — Normal conjugate mitosis (left) and diploidization with subsequent mitosis (right) in a dikaryotic tip cell of *A. cepistipes*. Dark area is nucleoplasm (chromatine), open circle nucleolus (Korhonen and Hintikka 1974).

process (Ullrich and Anderson 1978). In compatible pairings of haploid strains carrying complementary auxotrophic mutations, prototrophic hyphae are recovered at a high frequency from the periphery of the mated colony. The prototrophic tips invariably consist of uninucleate cells. The observed prototrophy is due to complementarity between the haploid, auxotrophic mates within a diploid nucleus. Diploids are occasionally formed also in sexually incompatible matings, but only at low frequency and only when strong selection is applied (Anderson and Ullrich 1982a).

Diploidy has also been shown by direct measurement of individual nuclear DNA contents using fluorescence photometry of material stained with the DNA binding fluorochromes mithramycin (Franklin and others 1983) and DAPI (Peabody and Peabody 1985) as well as of Feulgen-stained material (Peabody and Peabody 1984). In these kinds of studies, the fluorescence values of individual nuclei vary greatly because the vegetative hyphae are unsynchronized with respect to cell cycle, and because the technique inherently suffers considerable measurement error. Therefore, the most meaningful tests compare the average fluorescence values of similar cell types of distinctly different ploidy levels. In mithramycin-stained material, purified diploids from matings have on average twice the mean nuclear DNA content of their component haploid strains (Franklin and others 1983). Nuclei with DNA content consistent with diploidy are also found in mated single-spore isolates (Peabody and Peabody 1985).

The most convincing evidence for diploidy involved *A. ostoyae* and sexual reproduction. A single, uninucleate, putatively diploid cell was isolated from a mating of single-spore isolates. The resulting culture formed basidiomes, and all four segregant mating types were identified among the meiotic progeny (Guillaumin 1986a, Korhonen 1980).

Besides *Armillaria*, no other hymenomycete with a diploid vegetative stage is known to occur in nature. Exceptional diploid strains of *Schizophyllum* (Koltin and Raper 1968) and *Coprinus* (Casselton 1965) have been produced in the laboratory.

Somatic Haploidization

The diploid vegetative stage of *Armillaria* has proved to be remarkably stable. For example, of 1,224 hyphal tips isolated from 17 diploids resulting from both compatible and incompatible matings of auxotrophic strains, only two expressed segregant, auxotrophic phenotypes (Anderson and Ullrich 1982a). One segregant was from an A≠ B≠ diploid. It retained heterozygosity at both mating-type loci and expressed one of the two auxotrophic markers. The other segregant was from an A≠ B= diploid, and was no longer heterozygous at the *A* locus and expressed both auxotrophic markers. The mechanism of low-frequency "spontaneous" segregation is not known.

Another means of obtaining somatic segregants of *Armillaria* diploids was to use various agents known to cause somatic segregation in diploids of other, higher fungi. Of benomyl, ultraviolet light, formaldehyde, and para-fluorophenylalanine, only benomyl was effective in increased somatic segregation in *Armillaria* diploids (Anderson 1983). Two different kinds of selection can be used (Anderson and Yacoob 1984). When the parent diploid is crustose, colonies arising from fragments of benomyl-treated mycelium can be scanned for the fluffy morphology. Alternatively, when the parent diploid is prototrophic and heterozygous for auxotrophic alleles, colonies can be screened for auxotrophy.

The first method involves less labor because it is a visual screen. The second method involves individual testing of colonies by transfer to minimal medium. With these methods, a range of segregants can be obtained from diploids carrying various combinations of auxotrophic and mating-type markers. Some segregants retain heterozygosity at mating-type loci while some do not, and a variety of auxotrophic requirements are expressed in the segregants. Furthermore, the segregants have a variety of mean, nuclear DNA contents ranging from near haploid to near diploid levels (Anderson and others 1985). Because many of the segregants are no longer heterozygous at mating-type loci and have near-haploid DNA contents, the genetic segregation can be assumed to occur by haploidization during which one of each homologous chromosome is lost.

Overall, the parasexual system is a workable alternative to sexual reproduction for genetic analysis. This is especially so in *Armillaria* because some species/isolates of this genus do not fruit easily in pure culture. Benomyl-induced haploidization can also be used to obtain fluffy segregants from wild-collected diploid isolates (Anderson and Yacoob 1984). Haploidization may be useful, for instance, in cases when the species identification of diploid isolates in diploid-haploid matings proves difficult (Proffer and others 1987).

Benomyl's genetic effect on *Armillaria* diploids raises the possibility that the benomyl in isolation media Maloy 1974) might alter the *Armillaria* cultures recovered. Since the concentrations of benomyl used to inhibit common contaminant ascomycetes (Edgington and others 1971) are much lower than that required to destabilize diploids of *Armillaria* (Anderson 1983), however, we believe that low concentrations of benomyl can be safely included in media used to isolate *Armillaria*.

A Possible Haploid Stage in Basidiomes

A perhaps even more curious phenomenon than vegetative diploidy is the reappearance of the haploid stage in the basidiomes of most *Armillaria* species. As had already been shown by Romagnesi (1970), the subhymenial cells and the basidia of these species are clamped. Korhonen (1980) confirmed that these clamped cells are dikaryotic, and the cytophotometric studies of Peabody and Peabody (1985) showed that these paired nuclei have DNA contents consistent with haploidy. Korhonen and Hintikka (1974) obtained pure cultures of dikaryotic hyphae from young macerated gills. The dikaryotic cultures are unstable and rapidly change into monokaryotic diploid hyphae, just as do the dikaryotic hyphae from compatible matings. This characteristic differs among the *Armillaria* species. Among the European species, *A. borealis*, *A. cepistipes*, *A. ostoyae*, *A. gallica*, and *A. tabescens*, all have clamped dikaryotic basidia, whereas the basidia of *A. mellea* develop from diploid cells and are clampless (Guillaumin 1986a). As stated above, the dikaryotic stage is also not found in the compatible matings of *A. mellea*.

Concerning the non-European *Armillaria* species, Motta and Korhonen (1986) showed that the basidiomes of NABS VI are clampless (as are those of the corresponding European species *A. mellea*) while the basidiomes of NABS VII have clamped basidia, like *A. gallica*. According to Bérubé and Dessureault (1988, 1989), the American species *A. sinapina* (NABS V), *A. gemina* (NABS II), and *A. calvescens* (NABS III) all possess clamped basidia. In contrast, the five Australasian species *A. luteobubalina*, *A. novae-zelandiae*, *A. hinnulea*, *A. fumosa*, and *A. pallidula* have clampless basidia (Kile and Watling 1983, Podger and others 1978).

As *A. ostoyae* produces basidiomes easily in vitro, the hymenium cytology of the basidiomes obtained in pure culture could be observed by Korhonen (1980) and Guillaumin (1986a). Korhonen noticed that the basidia of *A. ostoyae* in pure culture were clampless and uninucleate (like the basidia of *A. mellea* in nature). Guillaumin (1986a) found that while a majority of basidiomes of *A. ostoyae* produced in vitro had clampless basidia, some did not. Even the same isolate sometimes yielded basidiomes with either clamped or clampless basidia, suggesting that the determining factor is environmental rather than genetic. The specific conditions determining the occurrence of clamped or clampless basidia, however, have not yet been identified.

The origin of dikaryotic elements in the basidiomes of *Armillaria* is as yet unclear. Tommerup and Broadbent (1975) observed that while the stipe cells are monokaryotic, dikaryotic hyphae arise from multinucleate cells near the developing gill folds of basidiome primordia. These authors also observed that the size of individual nuclei in the monokaryotic cells at the basidiome is about twice that in dikaryotic cells. These observations suggest that monokaryotic stipe cells, are diploid and that a nonmeiotic haploidization occurs in the basidiome trama which gives rise to haploid nuclei in the multinucleate cells and dikaryons of the gills. More recently, Peabody and Peabody (1985, 1987) reported that the monokaryotic cells of the stipe have a mean nuclear DNA content consistent with haploidy. The possible haploidization may thus occur at a stage earlier than proposed by Tommerup and Broadbent (1975).

While the nonmeiotic chromosome reduction presents an intriguing possibility, no precedent exists in other, higher fungi for such a regular, nonmeiotic reduction division occurring either within the basidiome or before basidiome initiation. Furthermore, because of the problems inherent in comparing the nuclear DNA contents of very different cell types, alternative explanations for the results of Peabody and Peabody (1985, 1987) are possible. First, one cannot assume that each individual cell contains a full DNA complement and that no DNA degradation occurred if the stipe cells are not known to be viable. Second, and perhaps less likely, the degree of DNA staining or of fluorescence quenching may depend on the specific cell type. These and other factors might produce a lower than expected average fluorescent yield for stipe cells as compared with other stages.

Whether the possible nonmeiotic haploidization occurs in the trama of the basidiome or at a stage preceding the basidiome formation, it would be expected to produce a mosaic of haploid strains including all four mating types from any diploid strain. If the stipe consists of a mixture of haploids, then, why do cultures isolated from the stipe invariably appear as typically crustose diploids? Arguably, mating may occur among haploid components of the basidiome isolated on artificial medium, but it should be possible to recover the haploid components by maceration or micromanipulation. To our knowledge, this has not been reported.

An alternative explanation for the origin of the subhymenial dikaryon is that no "extra" nonmeiotic haploidization occurs in the life cycle of *Armillaria* species, but that vegetative haploids may exist in the field along with diploids and may participate in the basidiome formation. Even if vegetative "germ-line" haploids do occur in the field, something must explain why cultures from vegetative material in the field usually appear crustose and diploid. Here, too, it could be argued that mating occurs among the haploid components

when the material is isolated into pure culture. If this is the case, then it should be possible to recover the vegetative haploids by maceration or micromanipulation.

Nuclear Behavior in the Hymenium

The behavior of basidium nuclei in *Armillaria* species has recently been investigated by Chahsavan-Behboudi (1974), Peabody and Motta (1979), Nguyen (1980), and Guillaumin (1986a). Two haploid nuclei enter the basidium of those species having a dikaryotic subhymenium, and one diploid nucleus enters the basidium of those species with a monokaryotic subhymenium. From this point, the overall pattern of meiosis and basidiospore formation appears to be similar to other hymenomycetes. The four nuclei resulting from meiosis migrate to four spores formed on the basidium. Various anomalies are frequently observed, however. Additional mitotic divisions may occur in the basidium, resulting in more than four nuclei. Only four nuclei, however, move to the top of the basidia and enter the developing basidiospores; the other nuclei degenerate. Also, the number of sterigmata can be two, three, or five instead of the usual four. A small number of basidiospores (1%-5%) are binucleate (Guillaumin 1986a). Observations of the basidia of *A. gallica*, *A. mellea*, and *A. ostoyae* suggest that the haploid chromosome number (n) in these species is four (Guillaumin 1986a, Nguyen 1980).

Identification and Occurrence of Biological Species

Identification

Since Korhonen (1978) and Anderson and Ullrich (1979), interfertility tests have become a common method for routine identification of species and for differentiation of unknown isolates into groups. Mating tests are performed using haploid tester strains (monospore isolates) that represent each species to which the isolate could possibly belong. The unknown isolate is paired with all the tester strains, and the mating reactions scored according to the appearance of the mycelium. The unmated haploid cultures are generally fluffy, and diploid cultures crustose. However, considerable variation may occur in colony morphology depending on the species, isolate, and culture conditions. Haploid cultures are sometimes rather crustose (especially in *A. gallica* and *A. cepistipes*); conversely, diploid cultures may be relatively fluffy, (especially in *A. mellea*). Furthermore, a diploid culture of some species often grows submerged in the agar medium without crustose mycelium (and aerial hyphae). In some species (*A. gallica* and *A. cepistipes*), the submerged mycelium discolors malt extract agar medium intensely brown; in others (*A. ostoyae*), it does not. On the other hand, the haploid isolates have a strong tendency for degeneration. Their surfaces become flat and wet, and they lose their ability for mating.

Distinguishing haploid and diploid cultures by appearance alone is not always possible. However, the distinction is usually clear-cut when the amount of aerial mycelium can be compared between pairings and unmated strains. The single best rule is that compatible matings show a reduction in the amount of aerial mycelium relative to the unmated strains, and incompatible matings show little or no reduction in aerial mycelium.

The safest identification in mating tests is obtained when single-spore isolates from the unknown specimen are used in the test (fig. 2.4). Because of the possibility that the tester and the unknown haploid culture may be conspecific but incompatible due to identical mating alleles, at least two different testers must be used for each species. The pairings are usually done on malt extract agar (1%-2%) in petri dishes. Because the diploidization process in *Armillaria* is rather slow, the

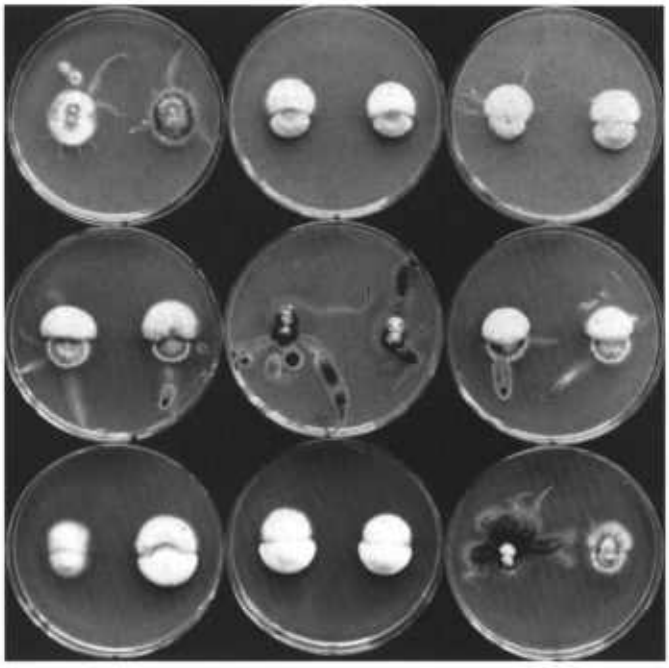


FIGURE 2.4 — Species identification of haploid isolates in a mating test. Each dish contains two pairings; in each pairing, the upper inoculum is a haploid tester strain, and the lower inoculum is the isolate to be identified. On vertical rows, there are two testers from *A. borealis* (sp. A), *A. cepistipes* (sp. B), and *A. ostoyae* (sp. C), respectively. On horizontal rows, three unknown haploid isolates have been paired with all six testers. The uppermost isolate proves to belong to *A. borealis*, the middle to *A. cepistipes*, and the lowest to *A. ostoyae*. Age of cultures: 20 days. (J. Anderson)

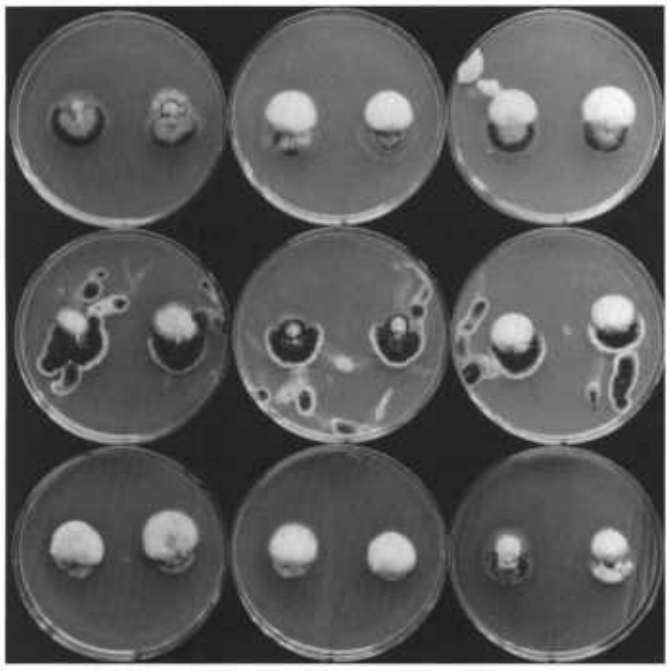


FIGURE 2.5 — Species identification of diploid isolates in a mating test. The arrangement is the same as in fig. 2.4, but the unknown isolates are diploid. Tester reactions like those of *A. ostoyae* (lowest right) are not uncommon; the testers show only slight inhibition in growth. (J. Anderson)

distance between the two inocula in each pairing should not exceed 3 mm. The results usually can be assessed after 3 weeks at room temperature, or earlier if the inocula are put closer to each other.

Diploid cultures can be identified in similar tests (fig. 2.5), which are analogous to the Buller phenomenon. Apparently because of diploidy, the testers' reactions in conspecific diploid-haploid pairings are usually much slower than in haploid-haploid pairings; sometimes the tester may fail to react at all. This sometimes makes the interpretation of diploid-haploid pairings difficult, and some patience is necessary for good results. According to our experience, a vast majority of diploid isolates can be safely identified in diploid-haploid pairings if six specific procedures are followed:

(1) This identification method should be used only in geographic areas where the species composition has first been investigated in haploid-haploid pairings. The unknown isolate must belong to one of the tester species.
(2) Use at least four haploid testers from each suspected species. The testers should be relatively fresh and not degenerate in colony morphology.
(3) Large tests containing material from several species are better than small ones. Always include unpaired "control" cultures of the testers and unknowns. It is also desirable to include known diploid cultures of different species in the test series for comparison.
(4) Read the results first after about 3 weeks (or earlier) and again after another 3 weeks or more. Spread many dishes on the table and compare the behavior of the testers in different pairings, especially in pairings with known diploids. Relatively small changes in the appearance and growth of the testers may be important. Do not necessarily expect a drastic change from fluffy to crustose.
(5) In haploid-haploid matings as well as in haploid-diploid matings, "black lines" are formed in the agar between the cultures if they do not belong to the same species. These lines can often help considerably in diagnosis. They should not be confused, however, with the margin of the pseudosclerotia consisting of aggregated ("bladder-like") cells (Mallett and Hiratsuka 1986).
(6) When the identification is unsuccessful in the first test, make a second attempt using a larger selection of testers from the suspected species.

Additional criteria may also help in identifying unknown diploid isolates, especially from European species. The morphology of mycelial mats in standardized pure cultures (i.e., on malt agar in petri dishes) sufficiently characterizes the species to assist identification (Guillaumin 1986a, Guillaumin and Berthelay 1981, Intini and Gabucci 1987, Mohammed 1987, Rishbeth 1986). The main drawback of the method is that it cannot distinguish *A. gallica* from *A. cepistipes*. Although the criterion of culture morphology is less helpful for identification of haploid cultures, mating tests alone are usually sufficient in this case. Guillaumin and others (1989a) have shown that the ability to reproduce in standard culture and the morphology and pattern of subterranean rhizomorph branching obtained in a mist box can also be used for identification (see table 1.3).

European Species

For the European *Armillaria* species, a complete synthesis between the concepts of biological and taxonomic species has been made. This means that the "biological species," which can also be regarded as taxonomic species, differ by many characteristics. Seven species of *Armillaria* have been found in Europe, five annulate and two exannulate. The fertility within each species and sterility between different species seem to be complete. *Armillaria mellea*, *A. gallica*, and *A. ostoyae* have a circumboreal distribution. Outside Europe, they have been found in North America and Japan. Interfertility seems to be almost complete between European and American populations of *A. mellea* (NABS VI) and *A. gallica* (NABS VII), respectively, but is only partial be-

tween these populations of *A. ostoyae* (NABS I) (Anderson and others 1980, Guillaumin 1986a). *Armillaria tabescens* may also exist in Europe, North America, and the Far East but recent matings between the European and American forms (Guillaumin unpubl.) indicate that they are intersterile. The situation is even more complex for *A. cepistipes*, a European species that appears to be partially interfertile with two different North American biological species, NABS V and NABS X (Anderson 1986, Anderson and others 1980), plus fully interfertile with NABS XI (=group F, Morrison and others 1985a). NABS XI will likely prove to be conspecific with *A. cepistipes*. NABS V, however, sufficiently differs from *A. cepistipes* to be described as a separate species (*A. sinapina*, Bérubé and Dessureault 1988).

Because a complete correspondence between the biological species and the morphological species of Europe has been established, many other kinds of data can complement or verify the results yielded by the mating tests (see chapter 1). Among these, the morphological criteria generally play the most important role, although physiological, morphogenetic, and biochemical characteristics may also be used (see chapter 1).

North American Species

In North America, identifying *Armillaria* species currently consists of placing unknown isolates in one of nine known (annulate) biological species. All but NABS IX an NABS X are now either formally equated to European species or are described as new species (Bérubé and Dessureault 1988, 1989). Since at least three North American groups, NABS VII, VI, and I, are probably conspecific with the European species *A. gallica*, *A. mellea*, and *A. ostoyae*, respectively, many properties of the three European species are likely to be found in their American counterparts. Nevertheless, such an extrapolation requires caution until more information on North American material is available. For example, Mohammed and Guillaumin (unpubl.) have observed differences between the European species and their American counterparts in such characteristics as culture morphology or the conditions needed for sexual reproduction in vitro. Moreover, within NABS I the isolates of eastern and western origin seem to differ in their ability to form basidiomes in vitro and in their level of interfertility with European *A. ostoyae* (Mohammed and Guillaumin unpubl.). Also, Mexican isolates of NABS I (*A. ostoyae*) have formed basidiomes in culture (Shaw 1989a). At present, we have no reason to believe that each *Armillaria* species is panmictic over its entire range. Even though each species is unique overall, genetic differences probably exist among geographically separated populations.

In addition to the species mentioned above, NABS II, III, IX, and X have been reported in North America (Anderson 1986, Morrison and others 1985a, Shaw and Loopstra 1988). Bérubé and Dessureault (1989) have formally described NABS II as *A. gemina* and NABS III as *A. calvescens*. NABS IX and X await further study. The original testers for all the North American biological species were from Anderson and Ullrich (1979; see also Anderson 1986). Several authors have used these testers to identify North American isolates by haploid-haploid pairings (Bérubé and Dessureault 1988, 1989; Dumas 1988; Mallett and Hiratsuka 1988; Morrison and others 1985a,b; Motta and Korhonen 1986; Proffer and others 1987; Shaw and Loopstra 1988). A large number of testers from these studies are now available. Morrison and others (1985a) discovered a new biological species, NABS XI. As some American groups are entirely or partially compatible with some European species, Motta and Korhonen (1986) and Guillaumin and others (1989a) could also identify some American isolates through matings with European testers. Wargo (1989) and Guillaumin and others (1989a) mated diploid isolates with the haploid testers (diploid-haploid matings) with less satisfactory results. In spite of recent progress, more information is needed before the breeding relationships of all *Armillaria* species in the Northern Hemisphere are known.

Australasian Species

Five *Armillaria* species have been found in temperate and subtropical Australasia. The situation is very similar to that of Europe after the studies of Kile and Watling (1981, 1983, 1988). The identification of Australasian *Armillaria* species is based on the morphology of the basidiomes. Mating tests have also been extensively used by Kile, who selected a range of haploid testers for *A. luteobubalina*, *A. hinnulea*, *A. novae-zelandiae*, and *A. fumosa*. The vegetative morphology of these species is somewhat different and can be helpful for identification. Four species form basidiomes in pure culture (Guillaumin 1986a; Kile and Watling 1981, 1983), which can also aid identification either through observation of basidiome morphology or by obtaining haploid mycelia.

Other Regions

Morphological species have been described from Africa, India, Central and South America, and the Caribbean (see table 1.1), but little is known about their status as biological species. Mohammed and others (1989) found genetic criteria of limited value in separating African isolates. Little is known about the situation in Africa, China, and southeast Asia.

Variation Within Biological Species

Because the present species concepts in *Armillaria* are relatively new, the variation within individual species is poorly understood. Relevant knowledge is accumulating rapidly, however. Casual observations suggest that intraspecific variation occurs in rhizomorph branching pattern, basidiome and vegetative morphology, pathogenicity, and physiological and biochemical characteristics. Given the variation with these parameters, it is not surprising that polymorphism in isoenzyme profiles (Lin and others 1989, Morrison and others 1985b) and restriction fragment patterns in nuclear (Anderson and others 1987, Anderson and others 1989, Anderson and Smith 1989) and mitochondrial DNA (Jahnke and others 1987, Anderson and Smith 1989) exist in *Armillaria* species as they do in other species of plants, animals, and fungi that have been investigated (see also chapter 1).

Perhaps the most intriguing polymorphisms occur at the mating-type loci. Although the total number of mating-type alleles has not been estimated for any *Armillaria* species, the numbers of alleles in small samples of strains from local environs in North America (Ullrich and Anderson 1978, Anderson and others 1979), Finland (Korhonen 1978), France (Berthelay and Guillaumin 1985), and Australia (Kile 1983b) have been determined. In all cases the number of alleles was on the order of 10. Considerably more alleles likely exist within each respective species over its entire range.

The Identification of Genotypes

The identification of fungal individuals (genotypes, clones) and the investigation of their spread in natural substrates may reveal valuable information about the ecology of the fungus in general and about its infection biology in particular. Three methods of genotype identification have been used in *Armillaria* studies. First, the identification can be done on the basis of cultural characteristics of the isolates (Rishbeth 1978b). Second, genotypes can be identified by "somatic incompatibility," the formation of demarcation lines in confrontations. In wood, for instance, the demarcation lines border the territories of different fungal individuals (Rayner and others 1984). Somatic incompatibility has been applied for the identification of *Armillaria* genotypes in several studies (e.g., Adams 1974; Anderson and others 1979; Hood and Morrison 1984; Hood and Sandberg 1987; Kile 1983b, 1986; Korhonen 1978; Mallet and Hiratska 1985; Shaw and Roth 1976; Siepmann 1985; Thompson 1984). The method is simple: two diploid isolates are paired in a petri dish and the confrontation zone is observed after a few weeks. When the mycelia from a local site are genetically identical, they intermingle in a pairing to a single homogeneous colony. When mycelia from a site are genetically different, they form a permanent demarcation line between each other in a pairing. The reaction can be intensified by cultivating the fungi in wood blocks (Hood and Morrison 1984).

The test based on somatic incompatibility is a very useful method for identifying fungal genotypes. Some reservations in its usefulness are necessary, however. It has been found in experiments carried out with several species of Basidiomycetes that this method does not always distinguish between closely related heterokaryons, especially sibcomposed heterokaryons (products of compatible matings between single-spore mycelia originating from one genotype) or their parent heterokaryon (Adams and Roth 1967, Barrett and Uščuplić 1971). In *Armillaria*, the situation is comparable: sibcomposed diploids, although genetically different, produce a distinct line of demarcation in only about half the pairings (Kile 1983b, Korhonen 1978). The occurrence of sibcomposed diploids in the neighborhood of an intensively sporulating parent mycelium is possible, at least, if not likely. Furthermore, the reactions between different diploid genotypes of the same species should not be confused with reactions between diploids of different species. In the latter case, the paired mycelia usually produce a black line along the demarcation zone. The black line is usually absent in pairings between two genotypes of the same species.

The most serious reservation about the use of vegetative demarcation lines for distinguishing strains is that the genetic basis for these vegetative reactions in *Armillaria* is not known. Because the intensity of the reaction varies among genetically different diploid strains, the reaction is probably determined by many loci with allelic variation. The demarcation lines are most useful as indicators of clonal identity when they are checked against other criteria (Kile 1983b, Korhonen 1978).

The third, and least ambiguous, method used in identifying *Armillaria* genotypes is the use of mating-type alleles as genetic markers (Berthelay and Guillaumin 1985; Kile 1983b, 1986; Korhonen 1978; Ullrich and Anderson 1978). Because many *A* and *B* alleles occur in the population, it is unlikely that two outbred diploids contain identical alleles. However, sibcomposed diploids and their parent mycelium always contain identical alleles. Using mating-type alleles as markers is considerably more laborious than using demarcation reactions because haploid cultures, and often a large number of matings between them, are necessary. More sophisticated methods, such as investigation of

isozymes or other protein spectra, and especially of nucleic acids, will undoubtedly open new perspectives for studies on intraspecific variation. For example, a recent study by Smith and others (1990) showed that several clones of *A. ostoyae* (NABS I) and *A. gallica* (NABS VII) in a local area each had a unique mitochondrial genotype that was stable during vegetative growth.

The Non-Heterothallic *Armillaria* Species

The African species *A. heimii* (synonym *A. fuscipes*, table 1.1) forms basidiomes easily in pure culture (Mohammed and others 1989). Monospore isolates of this species become crustose after 10-15 days in culture. When grown on an agar medium, they are identical to each other and also to the isolate (presumably diploid) that gave rise to the basidiome. Matings among a series of different monospore isolates from the same basidiome do not show any mating reactions. It can thus be assumed that *A. heimii*, at least in the conditions of artificial culture, is not heterothallic and tetrapolar as are the European, North American, and Australian species. Additional evidence for this difference in sexuality is that some monospore isolates have given rise to basidiomes that were morphologically identical to the basidiome from which the monospore originated. Monospore isolates from these first-generation basidiomes are also crustose and identical to each other, to the parent monospore, and to the original wild isolate. Again, no mating reactions can be shown among cultures of the same series.

Such a sexual behavior can be explained either by homothallism or by parthenogenesis. Cytological observations support homothallism: the basidia are clampless, the dikaryons are lacking, and each young basidium receives a single, large (presumably diploid) nucleus. However, the sequence of the nuclear divisions in the basidium is similar to that of the heterothallic species, indicating that meiosis (and not a succession of "normal" mitoses) occurs in the basidium.

Some other tropical *Armillaria* species from Africa or the West Indies could have a similar sexual behavior, according to the preliminary results of Mohammed and others (1989). The most plausible scheme for the life cycle of these tropical *Armillaria* species would be that the basidiospores are haploid and the young germinants convert to diploidy early. The remaining parts of the cycle would be diploid. However, the nuclei of the basidiomes of these species have not yet been studied by photometry.

The non-heterothallic behavior of the tropical species could affect their dispersal. The self-fertile spores of the homothallic species do not require mates in order to complete the life cycle, and therefore may be better colonizers than those of the heterothallic species.

The quite rare *A. ectypa*, a non-tropical *Armillaria* species which grows in arctic and alpine peat bogs of Europe, might also have a non-heterothallic behavior. It forms basidiomes easily in pure culture at 18°C. The monospore cultures from such a basidiome are identical to each other and, when paired, do not exhibit any mating reaction (Guillaumin unpubl.). The same is true of the monospore cultures isolated from basidiomes of natural origin (Korhonen unpubl.). Moreover, as with *A. heimii*, some single cultures are able to form basidiomes in vitro (Guillaumin 1973). In contrast with the tropical species, however, the basidia of *A. ectypa* are clamped and dikaryotic, whether the basidiomes are of natural origin (Lamoure 1965) or originate from in vitro culture (Guillaumin 1973). Thus, the life cycle of *A. ectypa* might be homothallic with a dikaryotic stage (the homothallic equivalent of a heterothallic species with a dikaryotic subhymenium like *A. ostoyae*) while *A. heimii* would be homothallic and lacking a dikaryotic stage (the homothallic equivalent of the heterothallic species with a monokaryotic subhymenium, *A. mellea*).

Conclusions

Genetic and cytological investigations of *Armillaria* have made reliable species identification possible, and demonstrated the value of the biological species concept for the genus. Moreover, recent studies have provided new information about the caryological cycles. The mating system of *Armillaria* species is generally tetrapolar, but the genus also contains homothallic species, especially from the tropics. The caryological cycle is exceptional in that *Armillaria* is the only hymenomycete known to have a persistent and widespread diploid vegetative stage in the field. Most species have a dikaryotic stage in compatible matings, but it is short and unstable with diploidization occurring in hyphal tip cells. Although vegetative diploids are very stable, benomyl will induce somatic haploidy. A phenomenon analogous to the Buller phenomenon is found between diploid and haploid mycelia of *Armillaria*, but its underlying genetic mechanism is unclear.

Despite the predominance of diploidy in the vegetative stage, the basidiomes of most species contain dikaryotic hyphae with clamp connections; the clamped basidia arise from dikaryotic cells. The origin

of this dikaryotic stage is still unclear. The basidiomes of other species do not contain dikaryotic hyphae, and the clampless basidia arise directly from uninucleate diploid cells. The adaptive consequences of caryological variation among different species remain unknown.

Although much recent progress has been made in understanding the genetic mechanisms in *Armillaria*, we see four areas that await investigation. First, with respect to life cycles, the nature and timing of the putative non-meiotic haploidization (if indeed it occurs at all) and the mechanisms of homothallism need to be resolved. We believe that appropriate experiments can help to clarify these aspects of the *Armillaria* life cycles. Second, because species and even individual genotypes can now be accurately identified, we can expect better resolution of epidemiological patterns, from long-range dispersal through local spread and infection in forests. Third, with sexual and parasexual crosses now available in the laboratory, it may even be possible to identify the determinants of pathogenicity. Finally, because of the considerable background on breeding relationships, morphology, ecology, and distribution of well-delineated species, *Armillaria* offers an excellent opportunity to use molecular characters to reconstruct phylogenetic relationships and to assess the relative roles of geographic isolation and intersterility in fungal speciation.

CHAPTER 3

Ontogeny and Physiology

Michael O. Garraway, Aloys Hüttermann, and Philip M. Wargo

The *Armillaria* life cycle, as with other members of the Agaricaceae, involves many developmental events which lead to the expression of several morphological forms. Specific structures include fruiting bodies or basidiomes, basidiospores, mycelia, pseudosclerotial tissue, and rhizomorphs. These structures enable *Armillaria* to accommodate various habitats and allow, directly or indirectly, various species and isolates to survive in the wild and to infect and colonize diverse hosts and substrates. This adaptability strongly influences the pathogenicity of *Armillaria* (see chapter 6), and we therefore discuss these structures and their development.

Structural differentiation and development in *Armillaria* are invariably preceded and accompanied by a series of intracellular changes which redirect metabolic pathways, redistribute organelles, and rearrange structural materials. Studies which would elucidate how differentiation and development are regulated in *Armillaria* would benefit microbiologists, ecologists, plant pathologists, and others who wish to control the survival, spread, and pathogenesis of this fungus. For reasons such as these, we review the nutrition and physiology of *Armillaria*.

As a root disease fungus, *Armillaria* is one of the most prominent killers and decayers of deciduous and coniferous trees and shrubs in natural forests, plantations, orchards, and amenity plantings throughout the world. Its roles include primary pathogen, stress-induced secondary invader, and saprophyte. Yet, the physiological bases for the varied roles are not well understood. Acknowledging this limitation, we discuss the physiology of the pathogen as it relates to host-parasite interactions.

The following presentations on *Armillaria* structures and their development, nutrition and physiology, and host-parasite interactions are intended to support the discussions of biology, ecology, and pathology in other chapters.

Structure and Morphogenesis

Armillaria resembles other agaricaceous fungi in the capacity of its hyphae to differentiate into various structures. Several of these structures enable this fungus to adapt to various environmental regimes and to exploit habitats and substrates which, without the structures, would be inaccessible. The structures in consideration include: (1) basidiomes, the main generative structure (fig. 3.1); (2) mycelia (fig. 3.2); (3) melanized cells (pseudosclerotia); (4) zone lines which *Armillaria* forms after interacting with other fungi and with tissues of infected hosts; and (5) rhizomorphs (fig. 3.3).

Development of Basidiomes

Descriptions of basidiome ontogeny in agaricaceous fungi, including an *Armillaria*, were given by Hoffman (1861). Later, Hartig (1874), Beer (1911), and Atkinson (1914) studied basidiome development in material identified as *A. mellea*. The latter two authors contradicted Hartig's observation on the developmental pattern. Fischer (1909a,b) studied *Armillaria mucida*, a species now placed in *Oudemansiella* (see chapter 1).

FIGURE 3.1 — Basidiomes of *Armillaria* (probably *mellea*) at the base of a dead red oak tree. (P. Wargo)

FIGURE 3.2 A — Mycelial fans of *Armillaria* (probably *calvescens*) on the root collar of a defoliated sugar maple sapling. (From Wargo and Houston 1974)

FIGURE 3.2 B — Mycelial fans of *Armillaria* at the base of a fumigation-damaged red pine tree. (P. Wargo)

FIGURE 3.3 — Rhizomorphs of *Armillaria gallica* on a white oak root. (P. Wargo)

Reijnders (1963) and Watling (1985) classified the basidiome developmental pattern of the few *Armillaria* species so far studied as monovelangiocarpic (only a universal veil encloses the hymenial primordium) as in exannulate species, or bivelangiocarpic (when the hymenium is enclosed by a partial and a universal veil) as appears to be the case in annulate species. Some of the latter species could possibly also be metavelangiocarpic (hyphae from various tissues proliferate to grow and cover the developing hymenium), but this remains to be established. Hymenophore development is probably ruptohymenial (differentiated from the background tissue) and the overall development pattern is stipitocarpic in which the young primordium is a stipe-like group or bundle (fascicle) of hyphae lacking an apical area of differentiated cells.

The most detailed morphological description of early basidiome development in *Armillaria* remains that of Atkinson (1914) for one of the North American species (see chapters 1 and 2). While Singh and Bal (1973) studied basidiome ultrastructure in an *Armillaria* sp., further work contrasting a wider range of *Armillaria* species and using modern morphological, cytological, and biochemical methods would advance our understanding of this differentiation which is essential for the completion of the *Armillaria* life cycle.

Production of Basidiomes in Culture

Molisch (1904) first reported the formation of *Armillaria* basidiomes in culture when he grew the fungus on autoclaved bread. Falck (1907) grew *A. mellea* from basidiospores to basidiomes and reported that light was required for basidiome development (Falck 1909). That basidiomes of several *Armillaria* species may be produced in vitro has been confirmed by many subsequent studies (Bothe 1928; Falck 1930; Fox and Popoola 1990, Guillaumin and others 1984, 1985, 1989a; Jacques-Felix 1968; Kiangsu Research Group 1974; Kile and Watling 1981; Kniep 1911, 1916; Lisi 1940; Long and Marsh 1918; Mańka 1961b; Raabe 1984; Reitsma 1932; Rhoads 1925, 1945; Rykowski 1974a; Shaw and others 1981; Shaw 1989a; Siepmann 1985; Tang and Raabe 1973; Terashita and Chuman 1987). These numerous reports, however, somewhat obscure the fact that basidiome production in vitro is not yet reliably achieved, although techniques are improving. This difficulty has been noted as an important limitation to some studies (Ullrich and Anderson 1978).

Many substrates have been found suitable for basidiome development. These include bread (Falck 1930; Kniep 1911, 1916; Molisch 1904); pieces of autoclaved wood or woodchips (Guillaumin and others 1989a, Molisch 1904, Raabe 1984; Siepmann 1985, Terashita and Chuman 1987); filter paper soaked in nutrients (Reitsma 1932); oranges (Guillaumin and others 1989a,

Jacques-Felix 1968, Shaw 1989a); maize kernels (Kile and Watling 1981); nutrient solutions or agars with various amendments including fruit or plant extracts (Kiangsu Research Group 1974, Mańka 1961b, Reitsma 1932, Rhoads 1925, Rykowski 1974a, Shaw and others 1981, Tang and Raabe 1973, Terashita and Chuman 1987). Basidiomes have apparently not been produced on a synthetic culture medium. While a complex carbohydrate source appears necessary to sustain mycelial growth and basidiome development, the role of inorganic nutrients, vitamins, or other compounds in stimulating basidiome production is poorly understood. Rykowski (1974) found that the fungicide sodium pentachlorophenolate at low concentrations stimulated basidiome development, a result confirmed by Shaw and others (1981).

Incubation conditions appear to affect in vitro development of basidiomes. Kile and Watling (1981) and Raabe (1984) noted that basidiome development in cultures coincided approximately with the natural basidiome season although other authors have not observed such an association (Rykowski 1974, Shaw and others 1981, Tang and Raabe 1973). However, most success seems to have been achieved when cultures are incubated in the dark after inoculation and then exposed to fluctuating temperature/light regimes (Guillaumin and others 1984, 1985, 1989a; Kiangsu Research Group 1974; Kile and Watling 1981; Rhoads 1925; Rykowski 1974; Terashita and Chuman 1987). While Tang and Raabe (1973) claimed light was not necessary for basidiome initiation, most authors conclude that both initiation and basidiome development require light (Rykowski 1974; Guillaumin and others 1984, 1989a). In this regard, *Armillaria* resembles other agarics (Lu 1974, Niederpruem 1963, Niederpruem and others 1964). However, significant scope exists to better define the light and temperature conditions that control basidiome initiation and maturation.

Some species of *Armillaria* appear to form basidiomes more readily in culture than others (Guillaumin and others 1984, 1985, 1989a; Rhoads 1925, 1945; Shaw and others 1981; Terashita and Chuman 1987). Apart from research by Guillaumin and others (1984, 1985, 1989a) using European *Armillaria* species, little comparative study has been undertaken of the basidiome development of different species under standard conditions, although Reaves (unpubl.) has produced basidiomes of NABS I, VII, IX, and X under standard conditions. Intraspecific variation in basidiome development also requires more quantitative assessment.

Pseudosclerotial Plates and Zone Lines

Since Hartig's first description, almost every paper on wood-destroying fungi or wood decay mentions or discusses the dark lines which are characteristic for wood degraded by fungi (for general reviews, see Bavendamm 1939, Rayner and Todd 1979).

These dark lines also form in wood infected by *Armillaria*. When wood is incubated under sterile conditions with a single isolate of *Armillaria*, zone-line formation can be obtained reproducibly within 2 months (Hansson and Seifert 1987), a process which is even considered as an economically feasible method to obtain special veneers (Hansson and Seifert 1987). The compartmentalization of decayed wood in living trees, first described by Falck (1924) and further elucidated by Shigo and his co-workers (Shigo and Tippett 1981), is a completely different phenomenon and will not be discussed here.

Campbell (1934) conducted the first systematic study on zone-line formation in wood decayed by *Armillaria*. He showed that zone lines can also form in sterile wood blocks. Since then, the physiology of zone-line formation has been studied by several authors, some of whom worked with *Armillaria*. They can be produced not only in wood blocks but also in sawdust cultures (Hopp 1938) and, during intra- and interspecific pairings of different isolates, in agar culture (Mallett and Hiratsuka 1986) or wood (Hood and Morrison 1984).

Three different mechanisms appear to promote pseudosclerotial plate or zone-line formation: mechanical and physical factors, antagonistic interaction of different mycelia (incompatibility reactions), and genetic factors within a species.

Mechanical and physical factors which have been suggested to induce pseudosclerotial plate formation include:

— fluctuating moisture content (Campbell 1934, Lopez-Real and Swift 1975, Radzievskaya and Bobko 1985a);
— gas phase composition (Lopez-Real and Swift 1977);
— wounding respiration-induced damage to hyphae (Lopez-Real and Swift 1977).

Incompatible reactions between vegetative mycelia of different species or different isolates of the same species resulting in the formation of black lines have been observed frequently on decayed wood (Radzievskaya and Bobko 1985b; Rayner and Todd 1977, 1979), and during pairings of different isolates in culture (see chapter 2).

Leslie and Leonard (1979) analyzed the genetics of injury-induced fruiting in *Schizophyllum commune* Fr. and found that mechanical injury may stimulate the formation of either pseudosclerotial plates or basidiomes.

The first (serendipitous) genetic analysis of zone-line formation was performed by Holt and others (1983). During their genetic analysis of basidiome formation in *Heterobasidion annosum* (Fr.) Bref., they found that zone lines were formed only in those crosses that also formed basidiomes.

Although some conflicting results remain when different studies are compared, the formation of pseudosclerotial plates is, in general, a genetically determined feature of many wood-destroying basidiomycetes which is induced by various external stimuli.

The morphological changes in hyphal structure caused by zone-line induction appear to be always similar regardless of the species, the mode of induction, or the substrate on which they are formed, either inside decayed wood or in culture (Hopp 1938, Lopez-Real 1975, Mallett and Hiratsuka 1986, Rayner 1976). The process of morphogenesis of pseudosclerotia can be divided into three distinct phases (Campbell 1934, Lopez-Real 1975): proliferation of hyphae, hyphal swelling and aggregation, and pigmentation and melanization of hyphae.

The pseudosclerotial plate of *Armillaria* is thus characterized by melanized, bladder-like cells which, especially in sawdust cultures, form a brittle plate. In such cultures, infrequently two types of rhizomorphs were produced (Lopez-Real 1975). Ribbon-shaped rhizomorphs were formed in deeper parts of the culture whereas round, pigmented rhizomorphs occasionally were generated directly from the surface crust. This association between the black crust and the pigmented rind of the round rhizomorphs indicates a close similarity between these two, differentiated structures (Campbell 1934, Lopez-Real 1975).

Rhizomorphs

Rhizomorphs and mycelial cords are examples of special morphological adaptations. They are discrete, filamentous aggregations which are formed by some fungi growing on the forest floor or, as in the case of the mycelial cords of *Serpula lacrymans* Pers.:F.S. Gray, even on concrete (Thompson 1984). Rhizomorphs differ from mycelial cords in that they are highly differentiated, are fully autonomous, and grow apically; typical mycelial cords are aggregations of parallel, relatively undifferentiated hyphae. In addition, rhizomorphs grow out from a food base into substrates that may not support their growth. This feature has been described for only one other fungus, *S. lacrymans* (Thompson 1984).

The capacity of certain fungi to produce rhizomorphs and cords confers several advantages (Thompson 1984). These include protection against deleterious external agents, translocation of resources, growth from a suitable food base into an environment which initially does not support growth, enhancement of inoculum potential, and amplification of individual hyphal sensitivity to external stimuli enabling directed growth responses.

Because of their frequency in some forest soils and their wide distributions, rhizomorphs had already attracted the attention of many mycologists by the middle of the nineteenth century. Moreover, because they were somewhat self-contained units they were described by taxonomists of that time as a separate fungus species: *Rhizomorpha fragilis* Roth. This species was further divided into two subforms, *R. subterranea*, which is found within soils, and *R. subcorticalis*, which grows beneath tree bark. An early description of the different forms of *R. fragilis* was published by Schmitz (1848). He is probably the earliest investigator to describe the remarkable stability of these structures and their ability to endure prolonged desiccation after which they appear to be dead, but revive when moistened. Schmitz inferred from observing rhizomorphs in rotted timber that the fungus was probably established in the trees before felling and utilized the timber as a food base following transfer to other locations such as mine shafts. He gives an "excellent description" of *Armillaria* rhizomorphs (quoted from Hartig 1874) and their effect on standing trees.

Like most of the leading mycologists of his time, Schmitz did not fully understand the cause-and-effect relationship between the occurrence of the fungus and the disease (Ainsworth and Sussman 1965, pp. 154-156; Hüttermann 1987). De Bary (1887, pp. 28-29) gives a record of the different views on the nature of rhizomorphs which were held at that time by such outstanding mycologists as Roth, Persson, deCandolle, Eschweiler, Acharius, Fuckel, Otth, Palisoth de Beauvais, Caspary, and Tulasne.

It was Robert Hartig who resolved these differences by providing decisive proof that the rhizomorphs found in forest soils belonged to the Honey Fungus (Hallimasch), *Agaricus melleus*, now known as *Armillaria* (Hartig 1874). He carefully observed the transition between the two rhizomorphic growth forms and prepared precise illustrations of this important morphological feature of the fungus. His suggestions that different environmental conditions and differences in availability of space, in either soil or beneath the bark of living trees, influence the development and morphology of the subcortical and subterranean forms of the rhizomorphs are still valid. His early observations that browning occurs only in rhizomorphs that have been exposed to air and not in those located under tree bark have been affirmed and explained in recent work, as has his observation that the browning

process, through the formation of a dense rind, inhibits further lateral growth.

Cytology of Rhizomorphs

De Bary (1869, 1887) presents a schematic drawing of mycelial aggregation and the resulting conspicuous form of a primitive *Armillaria* thallus (fig. 3.4). A much more detailed description of rhizomorph organization is given by Hartig (1870, 1874). He clearly described the organization of the thallus (fig. 3.5) with its three layers (cortex, subcortex, and medulla); and he described and illustrated the three forms of hyphae which are characteristic of these layers. He also observed the mucilagenous nature of the rhizomorph tip and the differential formation of the cell walls in the different layers of the rhizomorph. This work was followed by that of Brefeld (1877), who first described the apical growing region as a meristem. This view of rhizomorph morphology was not improved upon until methods of tissue preparation improved and electron microscopes were employed to study fungal structures. Motta (1969) examined thin sections of rhizomorph tips with the electron microscope and discerned the structure in more detail than Hartig or Brefeld were able to do (fig. 3.6). He confirmed Brefeld's earlier findings concerning

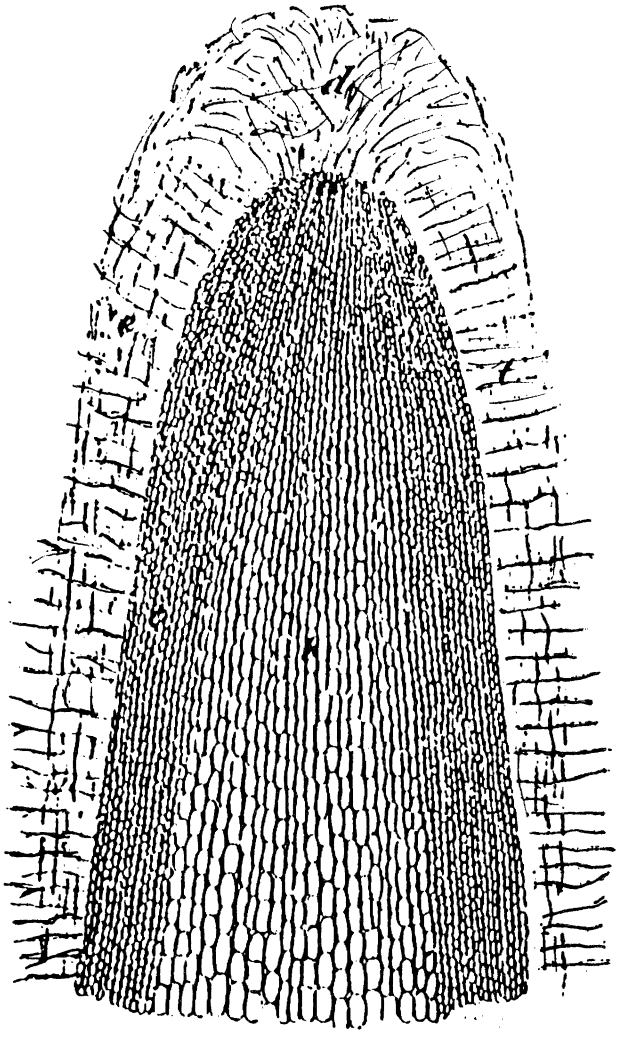


FIGURE 3.4 — Early drawing of rhizomorph (de Bary 1884).

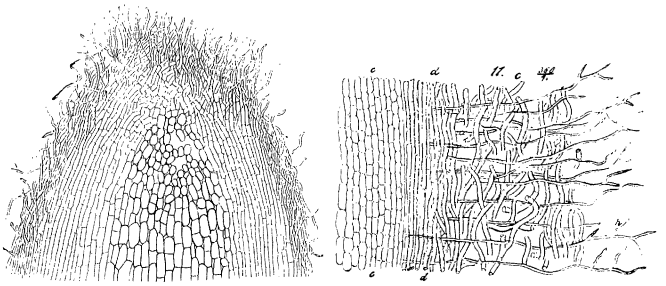


FIGURE 3.5 — Early drawing of rhizomorph (Hartig 1874).

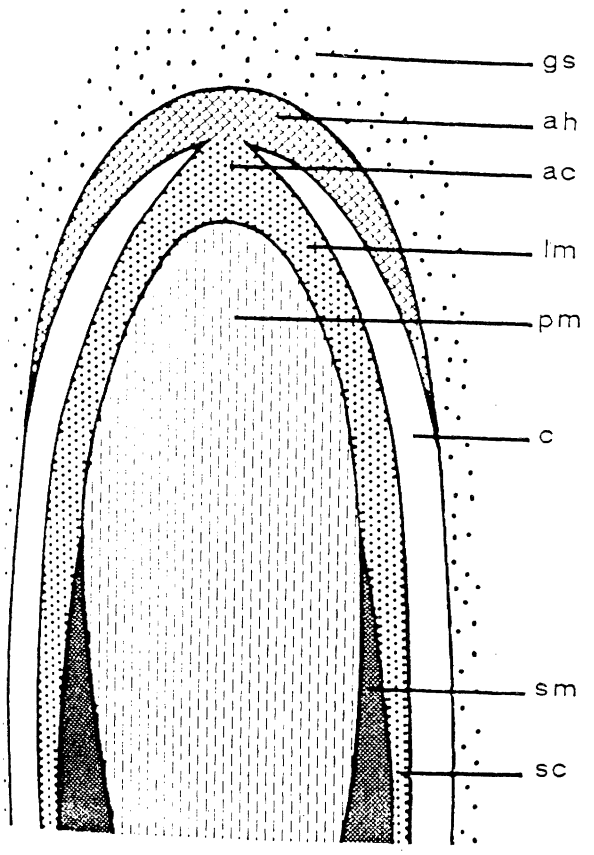


FIGURE 3.6 — Diagram of rhizomorph apex, illustrating the distribution of tissues and their origins: ah, apical hyphae; ac, apical center; lm, lateral meristem; pm, primary medulla; c, cortex; gs, gelatinous sheath; sm, secondary medulla; sc, subcortex. (From Motta 1969.)

the presence of a primary meristem in the rhizomorph apex. But he noted two types of meristematic activity: (1) the primary meristem that is located in the apical center, near the rhizomorph tip in which new hyphal elements are formed from apical initials; and (2) secondary meristems in the lateral regions of the apex where secondary cross wall formation takes place.

Differentiation of the apical initials involves synchronous nuclear divisions accompanied by segmentation in many planes. The apical initials are highly cytoplasmic, possessing non-membrane-bound fibrous bundles. Otherwise, they exhibit all the features normally found in hyphae of most basidiomycetes. The cells in the region assumed to be the primary meristem were shown by Motta to have dense cytoplasm with abundant ribosomes distributed throughout. Very few vacuoles were present and they were rather small. The number of nuclei per cell varied but could be quite high. The thickness of the initial wall remained constant during cell enlargement, indicating that the wall material was continuously synthesized in these cells. Schmid and Liese (1970) and Motta (1982) subsequently confirmed these findings.

Motta (1971) studied the histochemistry of the rhizomorph system. He found very high amounts of protein and nucleic acids, especially RNA, in the rhizomorph apical region. This discovery agrees with the view that this region is a true meristem. Large stores of glycogen were found in the cells adjacent to the meristem (i.e., apical center) and in the primary medulla (Motta 1971).

Wolkinger and others (1975) and Granlund and others (1984) studied rhizomorphs with the scanning electron microscope and discerned basically the same morphology described by Motta (1969) and Schmid and Liese (1970). Granlund and others (1984) used critical point drying which enabled them to better preserve the structure of the mycelia. This technique avoided hyphal collapse and allowed them to demonstrate that a loose network of hyphae (which they call the peripheral cover) covers the mature parts of the rhizomorphs. The method also enabled them to measure hyphal diameters in different regions of the rhizomorph and to calculate the resistance to solution flow through these hyphae (table 3.1). Obviously, from the values given in table 3.1 the hyphae of the medulla are the most likely candidates for solute flow-mediated transport in the rhizomorphs as was speculated by other previous authors.

Powell and Rayner (1983) studied the ultrastructural details of mucilage production by rhizomorphs. Using polyethylene bags to incubate rhizomorphs from logs of infected trees, they produced substantially larger rhizomorph apices with more clearly defined layers. The outer layers and the apical region were more densely packed with cells compared to those obtained by earlier studies. Their analyses of the morphology and formation of the mucilagenous layer confirmed the results of Hartig (1874), who described both long hyphae and swollen cells with a dense interior in the mucilage.

Mucilage was produced in tightly packed cells at the interface between the mucilagenous and cellular regions of the rhizomorphs. In this region, mucilage-containing vesicles coalesced with the plasma membrane, creating a mucilage-filled space between the membrane and all parts of the cell wall, with the septal plate being traversed by membrane-bound protoplasmic protruberances. After partial or complete digestion of the cell wall, this mass was released outside the cells.

Powell and Rayner (1984) found a specialized layer of cells, up to several cells wide, in the apical dome. These cells were biochemically very active as judged by their numerous mitochondria; and they were characterized by axial bundles of microfilaments, several of which occurred in each individual cell. These microfibril bundles were described earlier by Motta (1969). Powell and Rayner (1984) discussed the likelihood that this specialized layer of cells may provide a short-term supply of growth materials to the apical dome.

Some disagreement exists regarding the mechanism of rhizomorph growth which can be 19 mm or more per day. Brefeld (1877) and later Motta (1969) concluded that rhizomorph extension is due to a meristematic apical center containing actively dividing cells which give rise to the various other layers. This view was challenged by Rayner and others (1985), who suggested that rhizomorph extension might be analogous to the balanced lysis mechanism which has been proposed for

TABLE 3.1 — Diameter (μm), cross-sectional area $(\mu m)^2$, and calculated resistance to solution flow (μm^{-2}) of hyphae of different rhizomorph layers.

Tissue	Diameter	Cross-sectional area	Resistance to solution flow
Cortex	2.3	4.15	0.24
Sub-cortex	4.7	17.35	0.085
Medulla	13.9	151.75	0.0065

Source: Granlund and others (1984)

hyphal extension (Bartnicki-Garcia 1973). In this model, extension is possibly mediated by a plasticized apical dome which is driven forward by pressure generated within a tube with rigid side walls (rind) and compensated for by branching and growth of the intercalated apical hyphae. Rayner and others (1985) considered that plasticization could be facilitated by mucilage production which disrupts the continuity of the hyphal mesh that covers the dome. Rigidity could be achieved by melanization and compaction of the outer (rind) crust, and forward pressure could be provided via osmotically driven flow through the medullary region. However, too little evidence is available to support conclusively the hypotheses of Rayner and others (1985). Also, the basically filamentous organization of the rhizomorph apices might be obscured in thin sections of the dense cells of the apical dome (Schmid and Liese 1970). For example, compare the scanning electron micrographs obtained by Granlund and others (1984) with Motta's (1969) transmission electron micrographs of thin sections. We must conclude that the mechanisms underlying growth and extension of rhizomorphs are far from being completely understood.

Organization of the Differentiated Rhizomorph

All authors agree on the basic structure of the differentiated rhizomorph: the outer layer consists of mucilage and a loose network of hyphae surrounding a melanized and densely packed cortex. The cortex is the main structure which protects the rhizomorph in soil from being colonized by fungi and bacteria. Presumably, the melanin content of the outer cell walls confers the protection (Bloomfield and Alexander 1967, Khuo and Alexander 1967). Below the cortex lies the subcortical layer which forms the transition to the medulla. A loose mesh of wide-diameter hyphae, the medulla, is the main structure responsible for the transport of water and nutrients (Jennings 1984). Towards the center of the rhizomorph, the medullary hyphae become more and more loose, forming finally a central canal which is the main structure of oxygen translocation (Smith and Griffin 1971).

At the substrate-air interface, growing rhizomorphs can form "breathing pores" (Smith and Griffin 1971) that allow oxygen to diffuse through the intertwining hyphae into the central canal. These structures resemble buds of rhizomorph branches but have a completely different morphology. They are formed by tufts of hyphae, perhaps of aborted side branches, that have burst through the rind of the rhizomorph. The apices of these branches are composed of loosely intertwined hyphae with no organized meristem and are directly connected with the central canal.

Uptake and Transport of Nutrients and Water

The earliest studies on the nature and physiology of mycelial cords proposed a definite role for them in the uptake and especially the transport of nutrients and water (e.g., Falck 1912). The importance of rhizomorphs for transporting oxygen to growing parts of the fungus was first elucidated by Munch (1909), whose data were confirmed by Reitsma (1932). Schütte (1956) demonstrated that when fluorescein was applied to the base of rhizomorphs, it was transported to the tips. Morrison (1975) studied the uptake of radioactively labeled chloride and phosphate plus the uptake of ammonium ions. The two labeled ions were readily taken up by rhizomorph tips. When applied to their bases, these ions were translocated to the tips, but not in the opposite direction. The immersion of rhizomorph tips into a medium containing ammonium stimulated production of amino acids. Anderson and Ullrich (1982b) basically confirmed Morrison's observation that the transport in actively growing rhizomorphs is acropetal. Using C-14 labeled glucose and P-32 labeled phosphate as isotopic markers, they showed that diffusion was not a mechanism of transport. Only rhizomorphs living under aerobic conditions were able to absorb and to transport the nutrients, suggesting that the mechanism of transport is dependent upon aerobic respiration. Rhizomorphs living under anaerobic conditions were able to absorb the radioactive label but not transport to it.

Eamus and Jennings (1984) determined the water, solute, and turgor potentials in *Armillaria* rhizomorphs and found a considerable gradient of water and turgor potential from the tip to the base of the rhizomorphs. From these data and cytological evidence, the three criteria that Zimmermann (1971) said must be fulfilled for pressure-driven flow to be accepted as a translocation mechanism in plants are fulfilled in *Armillaria* rhizomorphs. These criteria are: (1) the conducting channel must be relatively impermeable to water in a lateral direction; (2) it must be very permeable to solutes and water in a longitudinal direction; and (3) turgor gradients must exist between source and sink. Eamus and others (1985) measured the internal structure and hydraulic conductivity of rhizomorphs. Their data support the view that long-distance transport occurs predominantly by solutes moving along vessel hyphae of the medulla. Granlund and others (1985) measured the velocity of translocation, estimating it to be 0.55-10.8 $cm.h^{-1}$; the flux of carbon and phosphate was 0.07-3.8[$nMcm^{-2}s^{-1}$]. They could not determine the chemical form in which carbon is translocated because of a rather vigorous lateral transfer, metabolism, and metabolic compartmentation of the label away from the stream within the rhizomorph. By changing the source-sink relations, they were able to demonstrate basipetal transport. In addition, bidirectional transport was observed.

The kinetics of phosphate uptake by rhizomorphs of *A. mellea* was studied by Cairney and others (1988). A biphasic mode of phosphate uptake indicated two different carrier systems with different Km and Vmax values. By chemically analyzing the homogenized rhizomorphs together with nuclear magnetic resonance studies of the intact system, they could discern between two orthophosphate pools, cytoplasmic and vacuolar, with most of the orthophosphate located in the vacuole. A significant portion of the cytoplasmic phosphorus was present in the rhizomorph as polyphosphate.

Concluding Comments on Rhizomorph Structure

Although development of *Armillaria* rhizomorphs has been studied for over 150 years, this process is still not well understood. Considerable work has been done on the structural and morphological features of rhizomorph development using both light and electron microscopes. But as will be evident later, virtually nothing is known about the biochemical mechanisms or genetic events that accompany their differentiation.

The morphology of rhizomorphs reveals a unique degree of differentiation. There are more than five types of tissues with different ultrastructures and functions in the organ. This makes rhizomorphs the most highly differentiated vegetative tissues of fungi, reaching almost the degree of differentiation of a plant root. The order and function of the different specialized cells and cell regions are summarized in table 3.2.

The picture that emerges so far is that of a highly differentiated organ with some specialization regarding solute transport and gas diffusion. Because of these structural features, *Armillaria* can grow in a hostile environment and compete with the microbiota in the forest floor. In addition, this structure enhances the pathogenic potential, including the capacity to enter the intact surfaces of a tree (Woeste 1956). It may also confer some competitive advantage over other root disease fungi, such as *H. annosum* (Shaw 1989b).

TABLE 3.2 — Specialized cells and regions of the *Armillaria* rhizomorph and their proposed functions.

1. Gelatinous sheet and mucilage layer at the apex:
   — protects the apex and facilitates its growth in the soil
2. Central region of the apex:
   — associated with mucilage production
   — includes a central meristem responsible for the growth of the rhizomorph
3. Circum-medullary cells of the apex:
   — provide a short-range supply of growth material for the apical dome
4. Lateral meristem:
   — originates lateral growth behind the apex
5. Melanized cortex:
   — the outer rind of the rhizomorph which protects it against fungal and bacterial attack, owing to its melanin content
6. Subcortical layer:
   — the secondary meristem associated with lateral growth
7. Medulla:
   — large cells associated with solute-mediated transport of nutrients
8. Breathing pores:
   — regions in the rhizomorph which facilitate oxygen uptake by the organ
9. Central canal:
   — a cavity within the rhizomorph which enables it to translocate gases

Nutrition and Physiology

In *Armillaria*, as with other fungi, factors that control growth and development of morphological structures may do so through the activation of key physiological and biochemical processes. Therefore, their appropriate manipulation may lead to the elucidation of underlying processes and mechanisms that determine growth and development. Since factors affecting growth and development of rhizomorphs and associated physiological and biochemical changes have been the focus of many physiological investigations of *Armillaria*, these topics will be emphasized. But because of the paucity of data concerning some aspects of *Armillaria* physiology, relevant research involving other fungi is included.

Garrett (1953) was the first to systematically study the induction of *Armillaria* rhizomorphs in pure culture on defined media. Working with agar plates, he showed that the production of rhizomorph initials is controlled by nutritional factors. Below we discuss *Armillaria* nutrition and physiology, including factors that affect rhizomorph development. We emphasize two themes: "factors" and biochemical changes affecting growth and development.

Factors Affecting Growth and Development

Nutritional Factors

Carbon Sources

Armillaria can utilize a wide range of carbon sources. This can be inferred from the reports of its wide host range (Raabe 1962a, 1979b; Rishbeth 1983; Singh and Carew 1983) and studies that show that some isolates

can utilize organic substrates for maintenance and growth in soil (Garrett 1960, Morrison 1982a) and on plant hosts (Rishbeth 1972b, Wargo 1980b). This view also is confirmed by the numerous reports that *Armillaria* can grow in culture on various carbon sources including carbohydrates (Wargo 1981a, Weinhold and Garraway 1966), lipids (Moody and Weinhold 1972a,b), phenols (Cheo 1982; Shaw 1985; Wargo 1983b, 1984), and alcohols (Weinhold 1963, Weinhold and Garraway 1966). The capacity of this fungus to fix CO_2 (Schinner and Concin 1981) suggests that this, too, may be a source of carbon for growth under certain conditions.

Despite the wide range of carbon sources they can utilize, *Armillaria* species seem to be selective in their ability to maximally utilize them for growth. For example, when glucose, fructose, and sucrose were compared, mycelia grew but were very sparse (table 3.3). This indicates that under these conditions these carbohydrates were used primarily as sources of energy for performance of vital functions and only sparingly for growth. In contrast, ethanol, added as a sole carbon source or as a supplement to a medium containing glucose, fructose, or sucrose, caused prolific growth of mycelia and rhizomorphs (table 3.3). Also, the fungus grew on ethanol-supplemented media containing glucose better than on fructose, which in turn was better than sucrose. Studies with C-14 labeled sugars suggest that these differences were partly related to different rates of uptake and utilization (Garraway 1975).

Examining the studies in which relative growth on various sugars was compared, one may conclude that *Armillaria* selectively utilizes carbon sources; glucose is the preferred carbohydrate. Moreover, when nutritional conditions change, the carbon source can shift from one which primarily maintains vital functions to one that both maintains these functions and supplies carbon for synthesis of compounds needed for growth and development.

As described later, such observations may help pathologists and ecologists interpret and explain certain in vivo aspects of *Armillaria* behavior. Presumably, when the interaction between *Armillaria* and a host is quiescent, there is limited access to host nutrients and growth promoters. Conversely, conditions associated with aggressive colonization of the host are likely to involve high access to host nutrients and growth promoters. Support for this view comes from studies such as those of Wargo (1972).

Nitrogen Sources

Besides a carbon source, *Armillaria* needs a suitable and adequate nitrogen source to grow and develop effectively. Garrett (1953) noted that *Armillaria* is not able to use nitrate as its sole nitrogen source. Also, although it grows on ammonium tartrate, the best growth was observed with amino acids. Similarly, Weinhold and Garraway (1966) studied how nitrogen sources affect growth and development of *Armillaria* in culture with glucose (0.5%) as a carbon source and ethanol (0.05%) as a growth stimulant. Casein hydrolyzate was the most effective nitrogen source followed by individual amino acids, several of which were more effective than inorganic nitrogen sources such as ammonium and nitrate (table 3.4).

TABLE 3.3 — A comparison of ethanol, glucose, fructose and sucrose, with or without an ethanol supplement, as carbon sources for mycelial growth and rhizomorph production by *Armillaria* in liquid culture.

Carbon Source (2.4 g/l)	Ethanol (.24 g/l)	Dry weight (mg.) Mycelium	Rhizomorphs	Total
Ethanol	-	22	18	40
Glucose	-	0.8	0.0	0.8
Glucose	+	26	20	46
Fructose	-	2.6	0.0	2.6
Fructose	+	8	8	16
Sucrose	-	0.9	0.0	0.9
Sucrose	+	6	4	10

Source: Weinhold and Garraway (1966)

TABLE 3.4 — A comparison of nitrogen sources for mycelial growth and rhizomorph production by *Armillaria* in liquid culture with ethanol (2.4g C/l) as carbon source.

Nitrogen Source (0.4 g N/1)	Dry weight (mg.) Mycelium	Rhizomorphs	Total
Casein	12	102	114
L-Aspartic acid	25	80	105
DL-Glutamic acid	13	89	102
L-Alanine	23	75	98
L-Asparagine	20	75	95
L-Glutamine	21	61	85
Glycine	35	36	71
DL-Leucine	15	46	61
$(NH_4)_2 HPO_4$	10	47	57
KNO_3	3	0	3
Control—no nitrogen	7	0	7

Source: Weinhold and Garraway (1966)

The effectiveness of casein hydrolyzate is related to its composition of mixed amino acids, including glutamic acid and leucine, which support vigorous growth of the fungus. Also, its effectiveness may be related to amino acid uptake which, in fungi, is governed by amino acid specific transport systems (Pateman and Kinghorn 1976). Transinhibition or transport system shutdown occurs as system-specific amino acids accumulate inside hyphae (Horak and others 1977). The variety of amino acids supplied by a substrate such as casein hydrolyzate would permit more transport systems to operate, resulting in a greater total nitrogen uptake. The capacity of a fungus to utilize the available nitrogen source is largely determined by the amount and type of carbon source. For example, Garrett (1953) noted that the optimal concentration of nitrogen to induce rhizomorphs increased as the carbohydrate concentration in the medium increased.

Rykowski (1976a) studied the interrelations between carbon and nitrogen levels in culture media on mycelial growth and rhizomorph production in several isolates of *Armillaria*. He found that at an appropriate nitrogen level, more carbon increased the mycelial dry weight. However, at a given carbon level, an increase in nitrogen above a certain level inhibited growth. Thus, the C:N ratio which varies for different isolates was found to be decisive for rhizomorph development.

Inorganic Nutrients

The requirements of *Armillaria* for inorganic nutrients are assumed to be comparable to those reported for other fungi. On this basis, relatively large quantities of magnesium, phosphorus, potassium, sulfur, and to a lesser extent, calcium may be required whereas copper, iron, magnesium, zinc, and in some instances, molybdenum may be required in minute quantities. These nutrients may play the same physiological roles in *Armillaria* as in other fungi (Garraway and Evans 1984). Although no systematic study has addressed the effects of various concentrations of these essential inorganic nutrients on *Armillaria* growth and development, Morrison (1975) recognized that the availability of inorganic ions affected its behavior in soil.

Vitamins

The importance of certain vitamins for growth was studied systematically by Garrett (1953), who compared the responses to thiamine and biotin. He noted that thiamine was required for growth but biotin was not. Also, Garraway (1966) noted that one isolate of *Armillaria* grew optimally in a synthetic culture medium supplemented with ethanol when the only vitamin supplied was thiamine. When this medium was deprived of thiamine, growth was reduced by 85%. In contrast, growth of this isolate was insensitive to either the presence of absence of biotin. Thus, except for thiamine, *Armillaria* appears to have the capacity, in common with many other decay fungi, to synthesize required vitamins from simple precursors (Garraway 1966).

Thiamine, as thiamine pyrophosphate, serves as the required coenzyme for several enzymes of intermediary metabolism that catalyze the removal or transfer of aldehyde groups. These include pyruvate carboxylase, transketolase, pyruvate dehydrogenase, and alpha-ketoglutarate dehydrogenase. Fungi are more often auxoheterotrophic for thiamine than for any other vitamin (Garraway and Evans 1984).

Organic Growth Factors

Several organic compounds produce rather dramatic effects on the growth and development of *Armillaria*. These compounds produce a response at concentrations substantially above those produced by typical vitamins, but far below those of nutrients such as carbon and nitrogen. Compounds which promote growth and development of *Armillaria* in this way include alcohols, auxin and related compounds, fatty acids, and phenols and related compounds.

Prior to 1963, optimal growth and development of *Armillaria* in defined media could be accomplished only by supplementation with undefined substrates such as yeast or figwood extract (Raabe 1962b, Weinhold and others 1962). In 1963, Weinhold discovered that low-molecular-weight alcohols and related compounds enhanced the fungus' growth and development (table 3.5). This made it possible to grow *Armillaria* on a completely defined medium and opened the way for critical studies on the nutrition and physiology of the fungus. Thus, in addition to being carbon sources, low-molecular-weight alcohols serve as organic growth factors in the sense described above. Growth was poor and rhizomorphs failed to develop on a synthetic medium containing glucose (0.5%) as the sole carbon source. But adding a small quantity (0.05%) of either ethanol, 1-propanol, or 1-butanol to the glucose medium stimulated prolific growth and rhizomorph formation (Weinhold 1963, Weinhold and Garraway 1966). Several other low-molecular-weight alcohols were shown to enhance growth and rhizomorph formation, but *Armillaria* isolates varied greatly in their ability to respond to different alcohols (Allermann and Sortkjaer 1973). These observations are of potential interest to those who study *Armillaria* ecology because soil microorganisms produce sufficient ethanol to promote rhizomorph development

TABLE 3.5 — Effect of ethanol-related compounds containing two carbon atoms, and other alcohols, in different concentrations, on rhizomorph production by *Armillaria*.

Conc. (mmole/ liter)	Length (cm) at 14 days*					
	Ethanol	Acetal-dehyde	Potassium acetate	Methanol	1-Propanol	1-Butanol
10.8	59.8+2.8	—	17.5+1.1	< 1.0	36.5+1.3	79.3+2.9
2.6	60.3+4.3	21.3+3.7	11.2+6.0	< 1.0	54.5+3.5	54.2+4.7
1.08	28.9+3.2	15.7+3.5	2.5+0.3		49.0+4.4	43.7+4.7
0.0	< 1.0	< 1.0	< 1.0	< 1.0	< 1.0	< 1.0

* Each value is the mean of at least six replications; standard error is indicated.

Source: Weinhold (1963)

(Pentland 1965, 1967); and ethanol may also be present in tree roots (Coutts and Armstrong 1976, Crawford and Baines 1977).

With the *Armillaria* isolate used by Weinhold, growth on a glucose medium supplemented with ethanol was equivalent to that on a medium containing ethanol (0.5%) as the sole carbon source (Weinhold and Garraway 1966). Analysis of the glucose culture medium at various times during the incubation period, however, showed that most of the growth occurred after the ethanol supplement was depleted from the medium (Garraway and Weinhold 1968b). This indicated that glucose was effectively used as a carbon source after a period of adapting to the ethanol supplement. When extra ethanol was added to a synthetic medium after 7 days (Garraway and Weinhold 1970) or 15 days (Sortkjaer and Allermann 1972) of incubation, the growth rate rose significantly. An increased growth-rate response to ethanol accompanied a decreased short-term uptake and utilization of glucose (Garraway and Weinhold 1968a, 1970) and an increased uptake of nitrogen and phosphate (Sortkjaer and Allermann 1973). Also, Sortkjaer and Allermann found that the rate of DNA and RNA accumulation increased as ethanol was added (fig. 3.7). These observations may provide clues to the mechanism(s) by which low-molecular-weight alcohols promote growth and development in *Armillaria*.

Several compounds with auxin activity promote growth and development of *Armillaria*. For example, synthetic media supplemented with 10 mg/l or more of indole-3-acetic acid significantly increased rhizomorph production (Garraway 1970, 1975). Also, 2,4-dichlorophenoxyacetic acid (2,4-D) stimulated the growth rate and amount of rhizomorphs produced by several isolates (Pronos and Patton 1979).

Such observations suggest that there is value in assessing models proposed to explain how auxins act on higher plants (Key 1969, Key and others 1967, Rayle 1973) to stimulate the growth of *Armillaria* rhizomorphs. The proposed response to auxin involving nucleic acid and protein synthesis might relate not only to the effects of auxin but to those of ethanol as well. According to this proposed mode of auxin action, the interaction of auxin with the plasma membrane releases a factor that moves through the cytoplasm and into the nucleus. The factor controls the activity of RNA polymerase in the nuclei and stimulates the synthesis of mRNA. The new mRNA is translated in the cytoplasm, resulting in new proteins which enhance cellular growth (Key 1969).

Lipids and fatty acids (Moody and Weinhold 1972a,b) and ortho- and para-aminobenzoic acid (Garraway 1970) strongly stimulate rhizomorph development when added to a defined basal medium. Since ethanol is linked metabolically to lipids and fatty acids (Garraway and Weinhold 1968a) and ortho- and para-aminobenzoic acids are linked metabolically to auxin, the possibility exists that all of these organic growth factors promote rhizomorph development by a common mechanism. Further molecular research will help to establish whether or not a common mechanism is involved in the response of *Armillaria* to these various growth factors.

Plant Extracts and Phenolic Substances

Many studies on *Armillaria* have reported that undefined media such as yeast extract or potato-dextrose-agar stimulate rhizomorph formation. Raabe (1962b) reported on the suitability of wood-based culture media for their stimulatory effect on rhizomorph induction. Also, Weinhold and others (1962) observed that a

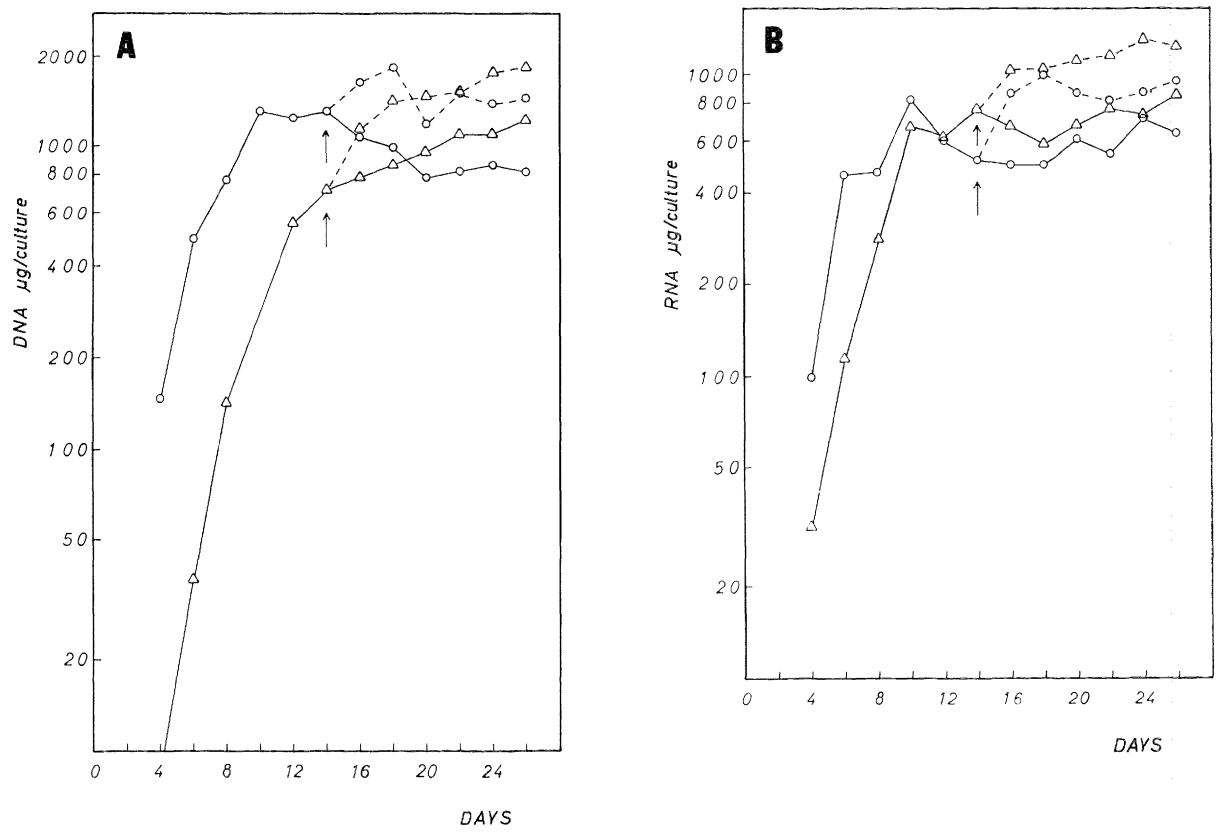


FIGURE 3.7 — DNA content (A) and RNA content (B) in *Armillaria* as a function of time following the addition of a boost of ethanol to culture media. The dashed lines show the content of DNA or RNA after the addition of extra ethanol; arrows indicate the time of addition of the extra ethanol. O = Diphenylamine method for DNA, orcinol method for RNA; Δ = UV method for DNA or RNA (from Sortkjaer and Allermann, 1973).

partially purified extract of figwood stimulates rhizomorph initiation and growth even though chemical analyses suggested that some factors other than ethanol or related compounds might be involved. More recently, Lin and others (1985) studied the induction of rhizomorphs by substances present in bark. Their observations that various plant constituents are able to induce rhizomorphs have been confirmed by more recent studies with auxins and phenolic compounds.

During the last decade, *Armillaria* has been reported to variously respond to phenolic compounds. Perhaps these studies received some impetus from earlier work which concluded that ethanol may enhance rhizomorph development by inhibiting glucose uptake and its conversion to phenolic inhibitors (Garraway and Weinhold 1970). The "phenol inhibitor theory" received added support when Vance and Garraway (1973) found that ethanol altered the phenol composition and lowered phenol concentrations in the fungus. Moreover, they noted that extracts of *Armillaria* thalli grown on glucose media had high levels of phenol and inhibited growth whereas extracts of thalli grown on ethanol-supplemented media had lower phenol levels and were non-inhibitory. This theory received further support when Oduro and others (1976) partially characterized from *Armillaria* a phenolic compound with antibiotic properties.

Elevated levels of certain phenols may stimulate growth and rhizomorph production whereas other phenols may be inhibitory. Thus, mycelial growth was enhanced by as little as 10 mg/l of shikimic acid (a precursor of phenol synthesis), protocatachualdehyde, and p-hydroxy benzoic acid (Garraway 1970). Also, guaiacol (Edwards 1981, Edwards and Garraway 1981), tannic acid (Cheo 1982, Shaw 1985) and substances rich in lignin (Guillaumin and Leprince 1979) promoted growth and rhizomorph development. But gallic acid, a derivative of oak bark tannin, inhibited certain isolates of *Armillaria* (Cheo 1982, Shaw 1985, Wargo 1980a). Although Armillatox, a proprietary phenolic fungicide, has been shown to inhibit rhizomorph development from wood blocks (Rahman 1978), it was ineffective as a control agent (see chapter 11).

In recent years, attempts have been made to use growth on phenol-amended culture media as an aid in distinguishing species and genotypes of *Armillaria*. Wargo (1980a) reported the reactions of several isolates grown on a gallic acid medium both with and without ethanol. He suggested that growth differences on gallic acid-amended media may indicate differences in pathogenicity or virulence of isolates. However, this method of testing pathogenicity was found to be unreliable (Shaw 1985). Efforts to distinguish *Armillaria* species according to their growth habits on culture media amended with phenolic compounds have been reported (Rishbeth 1982, 1986). Shaw (1985) found differences in the growth habits of 21 isolates of several *Armillaria* species depending on whether the phenol amendment used was gallic acid (the hydrolyzed form of tannic acid) or tannic acid. This could reflect differences in the permeability of fungal cell membranes to these phenols. Such differences could confound efforts to use phenol-amended medium as an aid to distinguish among species.

Environmental Factors

Growth and development of *Armillaria* involves a complex interplay of metabolic processes and other intracellular events. Therefore, environmental factors should help shape the expression of metabolic events leading to morphological changes. In the previous section, effects of nutritional factors on growth and development were emphasized. Below, we discuss effects of environmental factors such as temperature, aeration, pH, light, soil organic matter, and soil organisms.

Temperature

The earlier studies of Benton and Ehrlich (1941) and Bliss (1946) may have prompted the more recent systematic studies of the effects of temperature on mycelial and rhizomorph growth (Rishbeth 1968). Such studies provide information useful in predicting the fungal behavior on natural substrates and in soil. In this regard, Rishbeth (1968) noted the optimum growth rates of *Armillaria* mycelia and rhizomorphs on malt agar were 0.75 mm/day and 9.8 mm/day, respectively, at 28°C. The optimum growing temperature varied with the conditions but was about 22°C for rhizomorph growth from woody inocula through tubes of soil and for mycelial sheets growing along woody stems. Rhizomorphs produced by *Armillaria* isolates from different parts of the world grew maximally at 20°C and minimally at either 10°C or above 26°C (Rishbeth 1978a). How temperature affects field behavior of *Armillaria* is discussed in chapter 4.

Aeration

The vigor of *Armillaria* growth in soil and on natural substrates is related to aeration and, to a lesser extent, CO_2 levels. For example, the dry weight of rhizomorphs was reduced when the concentration of O_2 was lowered or that of CO_2 raised (Rishbeth 1978a). These studies and those of Ono (1970), Singh (1981b), and Morrison (1976) suggest that aeration strongly affects the distribution of rhizomorphs in soils (see chapter 4).

Smith and Griffin (1971) reported that oxygen affects both the rate of growth and the form of rhizomorphs. They acknowledged that maximum growth depends on high rates of oxygen diffusion within the rhizomorph's central canals. However, a partial pressure of oxygen of 0.04 atm on their outside surfaces inhibits rhizomorphs. They believed this occurred because high partial pressures of O_2 stimulated the fungus to produce p-diphenol oxidase, and that catalyzed the formation of a brown pigment in the rhizomorphs. This pigment overlays the walls of the cells and probably prevents growth by blocking the uptake of nutrients or the disposal of waste products by the cells.

pH

Benton and Ehrlich (1941) investigated how pH affects various *Armillaria* isolates in culture. The optimum pH for growth on malt agar was 4.5 at 21°C and 5 at 25°C. Studies with other fungi suggest that pH influences a fungus' ability to absorb various nutrients (Garraway and Evans 1984). Accordingly, the pathogenicity and aggressiveness that *Armillaria* exhibits on soils with low pH (Redfern 1978, Singh 1983) may be related to the pH effect on nutrient uptake by the fungus.

Light

Light inhibits vegetative growth of *Armillaria* (Weinhold and Hendrix 1963). Doty and Cheo (1974) found that mycelial and rhizomorph growth were inhibited by up to 80% when cultured in continuous light. Growth was reduced about 60% when cultures of the fungus were illuminated for 12 hr/day. Even exposure of only 2 hr/day inhibited growth by about 50%. The inhibitory effect of light occurred with several isolates. It was most inhibitory to isolates producing abundant rhizomorphs and less inhibitory to less productive isolates. Evidently, not all isolates or species of *Armillaria* are inhibited by light. For example, Benjamin (1983) showed that *A. limonea* produced rhizomorphs in the dark whereas *A. novae-zelandiae* would not produce rhizomorphs without light. This difference has been used as a diagnostic feature to separate isolates of the two species (Benjamin 1983, Hood and Sandberg 1987).

Growth of other rhizomorphic fungi appears to be inhibited by light. For example, Makambila (1978) noted that exposing cultures of *Rosellinia quercina* Hartig to light for 20 hr/day may inhibit rhizomorph growth up to 50%.

Soil Organic Matter

In vitro nutritional studies of *Armillaria* help validate the interpretation of field studies undertaken to evaluate the nutritional role of soil organic matter. Morrison (1976, 1982a) indicated that rhizomorphs absorb and utilize nutrients from soil and that soils rich in organic matter supply more nutrients for rhizomorph growth.

Effect of Other Organisms

Pentland (1965) observed that rhizomorph development was stimulated in pure culture by *Aureobasidium pullulans* (de Bary) Arnaud and attributed this effect to ethanol produced by this fungus (Pentland 1967). Also, Watanabe (1986) tested 121 fungal isolates for their ability to stimulate rhizomorph production either by co-culturing them with *Armillaria* or by amending *Armillaria* culture media with culture broth of the tester strain. He observed that 37 of the isolates tested effectively induced rhizomorphs. The most effective genera were *Macrophomina, Gliocephalis, Diploidia,* and *Sordaria* together with two unidentified species of Deuteromycotina. His reports did not include information on the chemical nature of the stimulatory factors involved.

Genetic Factors

Most researchers now acknowledge that species of *Armillaria* that occur worldwide comprise a complex of populations with distinctive genetic compositions (see chapters 1 and 2). Since genetic factors determine the expression of physiological and biochemical processes, genetic variation in *Armillaria* could be involved with reported cultural (Raabe 1966b) and pathogenic variations (Raabe 1967). Similarly, variation observed among *Armillaria* isolates in their responses to nutritional and environmental stimuli could be at least partially related to genetic differences. Examples cited previously include growth variation in response to low-molecular-weight alcohols (Allermann and Sortkjaer 1973), gallic acid (Cheo 1982, Shaw 1985, Wargo 1980a), and light (Benjamin 1983, Doty and Cheo 1974). However, nothing is known of the precise relationship between genetic control of responses to nutritional and environmental stimuli and the biochemical events involved. Also, the possible contribution of virus-like particles (Reaves and others 1988) to variation among *Armillaria* isolates should be considered. Chapter 6 provides further discussion of genetics in relation to pathogenicity and virulence.

Biochemical Changes Associated with Growth and Development

Voluminous literature relates biochemical changes to growth and development in fungi (Burnett and Trinci 1979, Moore and others 1985, Smith and Berry 1978); but the precise ways in which these changes regulate these phenomena are not known. However, studies of how biochemical changes relate to development in fungi provide clues to the regulatory mechanisms involved. A scan of the published literature suggests that many aspects of *Armillaria* biochemistry are either unknown or poorly understood. Therefore, formulating a good working hypothesis that implicates biochemical mechanisms in the pathogen's growth and development is difficult. We now focus on two biochemical themes that could have relevance to the regulation of growth and development of *Armillaria*: cell-wall polysaccharides and other macromolecules, and phenoloxidizing enzymes.

Cell-wall Polysaccharides and Other Macromolecules

Because cell walls control the shape of fungal cells and thalli, their composition and structure have been given particular emphasis in developmental studies. Ethanol, at concentrations that promoted growth and rhizomorph development, increased the incorporation of glucose into cell-wall polysaccharides by over 50% (Garraway and Weinhold 1968a, 1970). This could mean that cell-wall polysaccharide biosynthesis plays a part in the growth response (i.e., basidiome or rhizomorph formation) to various stimuli, as indicated in studies with other fungi (Stewart and Rogers 1978, Sietsma and Wessels 1977, Wang and others 1968, Wessels 1966). For example, the ratio of R-glucans (alkali-insoluble, highly branched beta-1,3- and beta-1,6 glucan) to S-glucans (alkali-soluble, alpha-1,3-glucan) was reported to change during basidiome development of *Schizophyllum commune* Fr. (Wessels 1965). Also, changes in cell-wall polysaccharide composition were correlated with genetically controlled changes in morphology in this fungus (Wang and others 1968). Moreover, cell-wall polysaccharide fractions from an *S. commune* mutant that failed to develop fully formed basidiomes were resistant to enzyme solubilization, whereas the same fractions from the wild-type isolate were soluble (Wessels 1966). Similar studies applied to *Armillaria* might help elucidate the role of cell-wall biosynthesis in its growth and development. A complex carbohydrate was recovered from mycelial cultures of *Armillaria* and some of its components have been char-

acterized (Bouveng and others 1967). But the importance for morphogenesis, if any, is not known.

Changes in large molecules not associated with the cell walls also occur during growth and development. For example, DNA and RNA contents of *Armillaria* increased at three times the rate of the dry weight in the first few days after ethanol was added to thalli (Sortkjaer and Allermann 1973). Also, similar increases in protein were observed in response to ethanol (Garraway unpublished). Thus, ethanol at concentrations which promote growth and development of *Armillaria* caused an early increase in constituents needed for nuclear division as well as for protein synthesis.

An association between lipids and growth and rhizomorph production in *Armillaria* was suggested from studies with C-14 labeled ethanol (Garraway and Weinhold 1968a). *Armillaria* preferentially incorporated ethanol into lipids. Furthermore, lipids of the type which are assumed to be present in *Armillaria* and its natural substrates, including lecithin, oleic acid, and linoleic acid, were able to replace ethanol as promoters of rhizomorph production (Moody and Weinhold 1972a,b).

Enzymes

Diverse enzyme studies have attempted to establish clues to the biochemical factors which regulate growth and development in fungi. Although changes in various enzymes have been reportedly correlated with morphogenesis, they are probably secondary to the more fundamental changes involved. This view is supported by studies involving enzyme levels and isoenzymes in *S. commune* (Bromberg and Schwalb 1978, Ullrich 1977). Work with *Armillaria* dehydrogenases are relevant in this regard. Mallett and Colotelo (1984) analyzed the activity and isoenzyme pattern of alcohol dehydrogenase during ethanol-induced rhizomorph formation. They found a significant increase both of the enzyme activity and the number of isoenzymes of alcohol dehydrogenases in the rhizomorphs but not in the mycelium. The relevance of the biochemical event studied appears obvious: alcohol dehydrogenase is needed for the metabolism of ethanol. But the relevance of this biochemical event to rhizomorph morphogenesis is still an open question.

Currently, some researchers are evaluating how the observed correlation between phenoloxidizing enzymes and rhizomorph development affects morphogenesis. The association of phenoloxidizing enzymes with rhizomorph growth received increased attention with the report that O_2 partial pressures above 0.04 atm at the rhizomorph surface enhanced accumulation of a brown pigment and inhibited its growth (Smith and Griffin 1971). Since high O_2 partial pressures stimulated the activity of p-diphenol oxidase, they proposed that the pigment formed as a result of enzymatic polymerization of phenols. Electron micrographs revealed that the pigment became localized in the intracellular spaces of the rhizomorphs. Smith and Griffin (1971) proposed that the pigment inhibited rhizomorph growth because an impermeable layer of polymerized phenol formed and it probably prevented the uptake of nutrients or the disposal of waste products by the cells.

More recently, Worrall and others (1986) have proposed a stimulatory role for laccase in rhizomorph initiation and development. Evidence supporting their claim includes several correlations. Ethanol and other substances that induced rhizomorphs in *Armillaria* also induced laccase (phenol oxidase) formation. In a range of isolates, rhizomorph production and laccase activities were positively correlated. Laccase was first detected just before the appearance of rhizomorph initials. Laccase activity peaked when rhizomorph growth was highest and fell to near zero when rhizomorph growth ceased. Laccase was not detected in cultures which were not induced to form rhizomorphs. Also, laccase activity and rhizomorph production, but not mycelial growth, were decreased by enzyme inhibitors with activity against laccase.

The contrasting interpretations of the role of phenoloxidizing enzymes by Smith and Griffin (1971) on the one hand, and by Worrall and others (1986) on the other, could involve different species of *Armillaria*. But contrasts are commonly encountered in *Armillaria* research. Edwards (1981) and Garraway and Edwards (1983) found that on a synthetic medium with casein hydrolyzate as the nitrogen source, a supplement of guaiacol (200 mg/l) promoted rhizomorph formation and increased phenoloxidizing enzyme (presumably laccase) activity. In contrast, when casein hydrolyzate was replaced with L-asparagine as the nitrogen source the same guaiacol supplement increased phenoloxidizing enzyme activity but not rhizomorph development. Adding an ethanol supplement to a medium containing guaiacol increased the activity of a laccase-like phenoloxidizing enzyme as well as rhizomorph growth. Thus, phenoloxidizing enzyme activity in *Armillaria* is apparently correlated with, but is not causatively related to, rhizomorph production in response to ethanol and other substances. Marsh and Wargo (1989) observed a similar association of laccase activity and rhizomorph formation among isolates of five species of *Armillaria*. Among the isolates that produced rhizomorphs, there was an association of higher laccase activity with greater rhizomorph production. Some iso-

lates, however, had laccase activity but produced no rhizomorphs (Marsh and Wargo 1989).

Phenoloxidizing enzymes have been implicated in the regulation of morphogenesis and differentiation of sporulating and resting structures in basidiomycetes and other fungi including *S. commune* (Leonard 1971, 1972, Phillips and Leonard 1976, Wessels and others 1985), *Coprinus congregatus* (Bull. ex St. Amans) Fr. (Choi and others 1987, Ross 1982), *Lentinus edodes* (Leatham and Stahmann 1981), *Podospora anserina* (Ces.) Niessl (Esser 1968, Molitoris and Esser 1971), *Sclerotium rolfsii* Sacc. (Chet and others 1972, Miller and Liberta 1977), and *Sclerotinia sclerotiorum* (Lib.) deBary (Wong and Willetts 1974). Very likely, they are important in these processes in *Armillaria* as well.

Nature of Phenoloxidizing Enzymes Produced by *Armillaria*

Because of the proposed causative association between rhizomorph morphogenesis and phenoloxidizing enzymes, the nature of these enzymes and their production by *Armillaria* needs to be reviewed. We do so giving consideration to the terminology for describing phenoloxidizing enzymes and the substrates used in their assay (Mayer 1987, Mayer and Harel 1979).

The commission on enzymes refers to monophenol monoxygenase (tyrosinase) as 1.14.18.1, diphenol oxidase (catechol oxidase, diphenol oxygen oxidoreductase) as 1.10.3.2, and laccase as 1.10.3.1 (Mayer 1987). This new classification differentiates between two reactions of the same enzyme, 1.14.18.1 for the cresolase activity and 1.10.3.2 for the catecholase activity of the same enzyme, catechol oxidase (Mayer 1987). Mayer proposes the general terms of "catechol oxidase" and "laccase" as the least confusing terms to use. Catechol oxidase can oxidize monophenols (tyrosinase or cresolase activity) or o-diphenols (catecholase activity); it cannot oxidize p-diphenols and this is diagnostic (Mayer and Harel 1979). Laccase can oxidize a wide range of substrates including mono-, di-, and triphenols. It can oxidize both o- and p-diphenols and its ability to oxidize p-diphenols is diagnostic (Mayer and Harel 1979). Catechol oxidase (tyrosinase) in fungi is primarily an intracellular enzyme and may have a role in melanin formation. Laccase is commonly excreted by fungi and has roles in lignin oxidation and degradation and detoxificiation of antifungal phenols in plant tissues (Mayer and Harel 1979).

Peroxidase (1.11.1.7) also catalyzes the oxidation of phenols by hydrogen peroxide (H_2O_2) and is non-specific for phenols. Much of the polyphenol oxidase activities reported in the *Armillaria* literature could include peroxidase activity if H_2O_2 commonly present in cell-free preparations was not removed. For example, Mallett and Colotelo (1984), using 4-aminoantipyrine, a substrate specific for peroxidase, detected peroxidase in exudates from *Armillaria* rhizomorphs. Also, they used catechol to detect phenol oxidase activity in the exudates. Since catechol is oxidized by tyrosinase, laccase, and peroxidase, a proportion of the phenol oxidase activity detected included peroxidase. These workers also noted the presence of beta-glucosidase, acid protease, and alkaline protease in the exudates.

Peroxidase activities were also reported in rhizomorph extracts of *Armillaria* by Lanphere (1934) and Lyr (1955). However, no substrate specific for peroxidase activity was used nor was catalase added to extracts to destroy H_2O_2 and eliminate peroxidase activity.

Both tyrosinase and laccase activities have been reported in mycelial extracts of *Armillaria* (Käärik 1965); but laccase can oxidize both tyrosine and guaiacol (p- and o-diphenols), the two substrates used. Both tyrosinase (catechol oxidase) and laccase activities were based on visual color development in tubes with agar and either guaiacol or tyrosine as substrates in the growth medium.

Stronger evidence for laccase (p-diphenol) activity was reported in rhizomorphs of *A. mellea* (Jacques-Felix 1968) and *A. elegans* (Smith and Griffin 1971), the latter now known to be *A. luteobubalina*. Worrall and others (1986), working with several *Armillaria* species, detected true laccase activity in culture liquid using 2,6-dimethoxyphenol and p-phenylenediamine as substrates. They found a general relationship of laccase production and species of *Armillaria* related to the proclivity of each species to produce rhizomorphs. *Armillaria mellea* isolates tended to have relatively high laccase activity and rhizomorph production, *A. ostoyae* isolates had low laccase activity and low rhizomorph production, and *A. gallica* had a broad range of laccase activities and rhizomorph production. No peroxidase activity was detected in these studies; however, only one of the isolates was screened for peroxidase activity (Worrall and others 1986).

Recently, Marsh and Wargo (1989) assayed phenol oxidases over time in three isolates from each of five biological species of *Armillaria*: NABS I (*A. ostoyae*), NABS III (*A. calvescens*), NABS V (*A. sinapina*), NABS VI (*A. mellea*), and NABS VII (*A. gallica*). Laccase (tetramethyl-benzidine=TMB=substrate) and peroxidase (TMB with and without catalase=substrates) were detected in extracts from mycelium and rhizomorphs and in the extra-cellular growth medium. Peroxidase activity was confirmed by the lowering of oxidase activity when H_2O_2 in the extract was destroyed by adding

catalase, and by assay with a substrate specific to peroxidase activity, aminoantipyrine. Peroxidase activity was not detected in all isolates, and a broad range of activities among the isolates with detectable peroxidase activity did occur. Tyrosinase activity (dihydroxyphenylalanine=L-DOPA= substrate) was found only intracellularly. They detected a general relationship of higher laccase activity with greater rhizomorph production among rhizomorph-producing isolates. However, laccase activity was also present in some isolates that produced no rhizomorphs.

Conclusions

The foregoing discussion of nutritional and environmental factors affecting *Armillaria* indicates that principles of fungal nutrition and physiology may be applicable to some aspects of its behavior in soil and on infected hosts. On the other hand, the discussion of biochemical factors that regulate growth and development indicates major information gaps for fungi in general and *Armillaria* in particular. More basic information at the molecular and biochemical levels is needed to develop a good working hypothesis to explain regulation of growth and morphogenesis in response to nutritional and environmental factors. When this information becomes available, more effective approaches to manipulating *Armillaria* in culture, in soil, and on its many hosts may be forthcoming.

Miscellaneous Themes in the Physiology of *Armillaria*

Protease

A protease with unique properties has been recovered from *Armillaria* (Broadbent and others 1972). This enzyme cleaves peptide bonds which are N-terminal to lysine residues in proteins (Hunneyball and Stanworth 1975, Lewis and others 1978). This specificity for lysine residues in the protein is maintained even when the positive side chain of the lysine is formylated and thus neutral in charge (Barry and others 1981).

The enzyme is very stable in the presence of denaturing detergents such as sodium dodecylsulfate. Because of this feature, the enzyme can be used to fragment proteins which are insoluble in water but can be solubilized by the addition of detergent (Barry and Doonan 1987). No information is available on the biological role of the enzyme. Whether it is secreted into the environment or present at unique points in the developmental cycle, such as during basidiome formation, is not known.

Antibiotics and Other Metabolites

In 1951, *Armillaria* was observed to exhibit considerable antibiotic properties when cultivated either on wood, solid media, or liquid media (Oppermann 1952). *Armillaria* antibiotics inhibited other fungi as well as bacteria. These findings were confirmed by Richard (1971). Later, Ohr and Munnecke (1974) found that the production of these antibiotics was considerably reduced when *Armillaria* was fumigated with sublethal concentrations of methyl bromide. The authors suggested that this is one reason for the effect of soil fumigation. It may predispose *Armillaria* to attack by biological control agents such as *Trichoderma* that would otherwise be restricted by the fungus' own antibiotics (see chapter 11).

The chemical nature of the antibiotic substances and other metabolites produced by *Armillaria* was elucidated in subsequent years by several groups of scientists. Oduro and others (1976) isolated four chloroform-soluble substances for which antibiotic activities were determined by bioassays with either *Bacillus* sp. isolated from fumigated citrus roots naturally infected by *Armillaria* or cultures of *Cladosporium cucumerinum* Ellis and Arth. The authors were able to show that antibiotic activity was produced by all 17 *Armillaria* isolates used.

Detailed studies by several authors (Ayer and MacCauley 1987, Donnelly and others 1982, Jungshan and others 1984, Midland and others 1982, Obuchi and others 1990) have revealed that various isolates of *Armillaria* have at least 10 different compounds with antibiotic properties. Two aspects of the chemical nature of these substances are rather interesting. First, they are mostly complicated sesquiterpenoid esters, some belonging to the protolludane group. The organic acid to which they are bound is, suprisingly, the same substance which has been identified as the antibiotic substance of *Sparassis crispa* Wulf.: Fr. (Falck 1907, 1909, 1924, 1930). Second, these compounds contain a rather simple aromatic, Sparassol or orsellinic acid, which in all tests exhibits high antifungal and antibacterial activity (Cwielong 1986). Apparently, *Armillaria* uses the same chemical weapon as does *S. crispa* with the modification that sesquiterpenoids are attached to the aromatic group. Thus, the *Armillaria* antibiotics would penetrate more easily through membranes and would probably be more toxic than the unsubstituted Sparassol.

The variety of antibiotic substances produced by *Armillaria* and their high toxicity against microbes may explain, in part, why this fungus is so successful in its natural habitat and also some of its medicinal properties. For example, folklore of early American loggers

tells of woodsmen who would wrap their wounds from accidental cuts in an *Armillaria* fan. This protected them from further irritation and enhanced healing. Also, tablets containing artifically cultured mycelia of *Armillaria* are used in China for treating of dizziness, headache, neurasthenia, insomnia, numbness in limbs, and infantile convulsions (Jungshan and others 1984).

Bioluminescence

Bioluminescent fungi have interested biologists for some time (Glawe and Solberg 1989). More recently, attention has been given to the biochemical mechanisms involved (Airth and others 1966, Danilov 1987).

Armillaria is one of several bioluminescent basidiomycetes (Guyot 1927). Airth and Foerster (1960) prepared a self-portrait of a 15-day culture of *Armillaria* that showed high luminescence in the peripheral region (young cells) and less in the central area (older cells). A similar, more precise study using photomicrography and a different species of *Armillaria* (Berliner and Hovnanian 1963) showed light emission occurred throughout the entire cell.

The characteristics of the light emitted by *Armillaria* and other fungi have been investigated. Airth and Foerster (1960) noted the emission maximum of 528 nm was similar to that of other fungi but different from that of bacteria. They found that the energy of activation for emission in *Armillaria* is 17,500 calories with a temperature optimum of 26°C. Berliner (1961) suggested that fungi which exhibit bioluminescence may emit some waste energy of oxidation as light instead of heat. Also, Berliner noted that *Armillaria* took a longer time than other fungi studied to attain maximum light emission values, but sustained luminescence of 10 weeks equaled or exceeded that of other fungi.

Effect of Environment, Nutrition, and Growth Factors

The effects of temperature, exposure to X rays and ultraviolet light, nutrition, and growth factors on luminescence in *Armillaria* and other fungi have been reported.

Temperature

Light emission was low at -10°C and low or non-existent above 40°C (Airth and Foerster 1960) with the optimum temperature in the range of 18-26°C. Berliner (1961) noted a similar optimum temperature for light emission by several basidiomycete fungi including *Armillaria*.

Ultraviolet and X-irradiation

Ultraviolet irradiation inhibited light emission from *Armillaria* and other fungi (Airth and Foerster 1960, Berliner 1963, Berliner and Brand 1962). The effects observed varied with the wavelength of incident radiation, the time elapsed, and the fungal species used. In contrast, X-irradiation enhanced luminescence from *Panellus (Panus) stipticus* (Bull: Fr.) P. Karst. (Berliner 1961) and probably would produce a similar effect on *Armillaria*.

Nutrition

The relationship between light emission and nutrition has been reviewed (Harvey 1952). Airth and Foerster (1965) reported a specific pH and nitrogen source for optimum light emission by *Collybia velutipes* (Fr.) Sing. On this basis, optimal nutritional conditions for maximum light emission presumably exist for *Armillaria* as well.

Growth Factors

Luminescence in *Armillaria* responds to growth factors according to the concentration and type of factor used. For example, the light output was intensified more than 150% when *Armillaria* was grown on a medium containing 0.75 mg/l of biotin. Also, kinetin at 0.25 mg/l increased light output, but 6-benzylaminepurine had no effect (Berliner and LaRochelle 1964). The effects of antibiotics on light emission have also been studied (Berliner 1965).

Mechanism of Fungal Bioluminescence

Studies with *Armillaria* and other fungi have identified the key biochemical steps involved in fungal bioluminescence. For example, Airth and Foerster (1962) presented evidence that fungal bioluminescence involves the following:

(a) either reduced nicotinamide adenine dinucleotide (NADH) or reduced nicotinamide adenine dinucleotide phosphate (NADPH);
(b) an electron acceptor found in hot water extracts;
(c) soluble dehydrogenases;
(d) molecular oxygen;
(e) the particulate enzyme luciferase.

The proposed reaction involved in light emission is:

$$2NADH + X \xrightarrow[\text{enzyme}]{\text{soluble}} XH_2 + 2NAD^+$$

$$XH_2 + \tfrac{1}{2}O_2 \xrightarrow[\text{luciferase}]{\text{particulate}} X + HOH + \text{light}$$

The similarities and differences of light emission between fungi and bacteria have been noted (Airth and others 1966). However, fungal and bacterial bioluminescence and chemoluminescence may have close links not only in their physical nature but in their biochemical nature as well.

Physiology of Host-Pathogen Interactions

Understanding the physiological bases for pathogenesis and the interactions of *Armillaria* species with their hosts is the key to understanding the variation in pathogenicities among and within the species of *Armillaria* that we now know. Unfortunately, much of the work that has been conducted in this area lacks essential taxonomy of the fungus. Results of these studies, therefore, may reflect the physiology of a single species, one or several genotypes within a species, or several different species all interacting with hosts that may or may not be resistant. Our current understanding, and hence what is presented herein, of what stimulates and controls penetration and colonization of a substrate by *Armillaria* is incomplete for any single species. What we know is probably a composite of several different *Armillaria* species interacting on susceptible and resistant hosts.

Genetic Control

The infection processes, resistant reactions, pathogenicity and virulence, and disease development within the host tree are discussed in chapters 4, 5, and 6. These processes represent host-pathogen interactions and involve the physiology of metabolic regulation of the fungus and host. Metabolic control of these interactions is determined by the genetic control of the physiological processes as modified by the environment (Daly 1976).

The reaction of host and fungus, therefore, depends on the host species that is attacked, the species and perhaps genotype of *Armillaria* that is attacking, and the environmental conditions under which host and fungus are growing. Most historical information on host-pathogen interactions focuses on the differences in response among host species. Little attention has been paid previously to differences in the pathogen since it was considered for the most part to be a single species. Now that several species of *Armillaria* are recognized with different pathogenic capabilities on different hosts (Davidson and Rishbeth 1988; Rishbeth 1982, 1985b), previous reports on host-pathogen interactions must be re-examined.

The infection process is both mechanical and enzymatic. Since penetration of the outer bark is reportedly similar in both the susceptible and the resistant reactions, subsequent colonization of the inner bark and cambial zone tissues differentiates the susceptible from the resistant reaction (Thomas 1934). These observations are based on reactions of hosts with single isolates of unknown species of *Armillaria*, although some attempts have been made to assign species names to some isolates used in these historical studies (see chapters 4 and 6). Whether all species of *Armillaria* can successfully penetrate the outer bark is not known. Wounding of the roots can enhance infection by *Armillaria* (see chapters 4 and 7), and perhaps some species of *Armillaria* are unable to penetrate intact bark.

Metabolic Control

Little work on the metabolism of *Armillaria* species in association with their hosts has been conducted. Therefore, mostly metabolic capabilities of *Armillaria* and their potential for interacting with hosts are reported here.

Pathogen Factors

Suberinase

Bark apparently offers limited resistance to penetration by *Armillaria*. Even periderms formed in response to the penetrating hyphae are unable to contain its growth (Rykowski 1975, Thomas 1934). The fungus can apparently grow faster than developing periderms and invades around them (Rykowski 1975) or penetrates directly through the periderms, probably by enzymatic activity (Arthaud and others 1980, Rykowski 1975, Thomas 1934). *Armillaria* can degrade suberin. Swift (1965) reported that the fungus, grown on ground bark of *Brachystegia spicaeformis*, caused a 59% loss in suberin content of the bark. *Armillaria* also produced hydrolytic enzymes when grown for 10 months on 0.5% raspberry suberin medium supplemented with salts, thiamine, and ethanol (Zimmermann and Seemüller 1984). Concentrated enzyme preparations from culture fluids caused up to 1% dry weight loss of suberin preparations after 16 hr incubation. Gas chromatographic analyses of the released material indicated that the components constituted a major part of the aliphatic monomers present in suberin (Kolattukudy and others 1981). How important suberin degradation is in the infection process is uncertain.

Polyphenol Oxidases

Armillaria produces phenol oxidases during the infection process. Discoloration, especially browning of

tissues, has been observed commonly during the infection and colonization process (Rykowski 1975; Thomas 1934; Wargo 1977, 1984a). Discolored bark in advance of colonized bark in black and white oaks had significantly less total phenols and more oxidized phenols than contiguous or noncontiguous healthy bark (Wargo 1984a). In colonized bark, total phenols were only 22% and 46%, respectively, of that in healthy bark of black and white oaks; and oxidized phenol levels were 3 and 3.5 times greater than in healthy bark (table 3.6). Phenol levels in discolored bark from wounded only bark tissues were also lower after 4 weeks than in healthy contiguous bark, but not as low as in colonized bark. Levels of oxidized phenols in discolored bark from wounded-only tissues did not increase as much as in colonized tissues.

Oxidation of the phenols in root tissues can result from both fungal and host polyphenol oxidases. No reports distinguish between host and fungus-mediated phenol oxidation. Fungal enzymes can oxidize phenols as a result of separate or combined effects of peroxidase, tyrosinase, or laccase depending on the phenolic substrates. *Armillaria* possesses all three enzyme activities and peroxidase and laccase can be secreted to oxidize phenols extracellularly, as described previously in this chapter.

Very limited information details the role of phenoloxidizing enzymes in the pathogenic process. Marsh and Wargo (1989) screened three isolates each of *A. ostoyae, A. calvescens, A. sinapina, A. mellea,* and *A. gallica* for production of constitutive phenol oxidases. Many, but not all, of these isolates were rated by other researchers in pathogenicity studies. The pathogenicities of the remaining isolates were rated by Marsh and Wargo as high, moderate, or low, based on their association with the host tree from which they were isolated. No obvious correlations of constitutive enzyme levels with pathogenicity were detected.

Phenols and other host substances can inhibit hydrolytic enzymes of fungi, thus restricting their activities on host cell walls and membranes and preventing infection and colonization. Polyphenol oxidases cause the oxidation and polymerization of compounds that are potentially toxic to the fungus, allowing infection and colonization to proceed in tissues rich in phenols. This reaction is apparent at the leading edge of mycelial fans colonizing living tissue. Here, an advancing band of oxidized (browned) tissue precedes the advancing mycelium (fig. 3.8). There is some evidence that these brown pigments induce wilt in infected plants. Thornberry and Ray (1953) isolated a dark brown protein-like pigment produced by *Armillaria* in liquid me-

TABLE 3.6 — Changes in mean concentrations of soluble phenols and their oxidation products effected by *Armillaria* in bark of roots of black and white oak trees naturally colonized by the fungus.

Species and tissue state	Phenols[1] total	Tannins[1]			Phenols[2] oxidized
		Total	Hydrolyzable	Condensed	
		mg phenols/g tissue			
Black Oak					
Healthy, control	167a	128a	143a	13a	238a
Healthy,contiguous	161ab	124ab	136a	13a	243a
Discolored	145b	107b	61b	11a	306a
Colonized	37c	31c	22c	8b	731b
SE	±5	±5	±3	±2	±30
White Oak					
Healthy, control	196a	147a	147a	15a	352a
Healthy,contiguous	170a	136a	160a	8b	621ab
Discolored	158a	124a	107b	9b	742b
Colonized	90b	67b	63c	11ab	1235c
SE	±10	±10	±8	±2	±80

Source: Wargo (1984a)

[1]Total phenol and total and hydrolyzable tannins - mg tannic acid equivalents/g freeze-dried bark: condensed tannin - mg catechin equivalents/g. Mean of 7 observations. Significant differences by ANOVA and Tukeys studentized range test (P≤0.05) indicated by different letter.

[2]Absorbance of solutes from 100 mg bark in 10 ml water at 450 nm and 1 cm light path used as estimate of oxidized phenols.

FIGURE 3.8 — Discolored brown zone in both bark and wood in advance of the mycelium. Note rhizomorphs on surface of primary root. (P. Wargo)

FIGURE 3.9 — Advanced decay of root wood by *Armillaria* (also note discolored brown zone in advance of mycelium). (P. Wargo)

dium. The pigment induced wilt in tomato seedlings and peach twigs at low concentrations. There is, however, no evidence that this mechanism operates in large mature trees.

These phenoloxidizing enzymes are also important in wood degradation (fig. 3.9). *Armillaria* is classified as a white-rot fungus because it degrades and removes the lignified material from the cells, leaving the white cellulose and hemicelluloses somewhat intact (Campbell 1931, 1932). Campbell also found that decay of wood by *Armillaria* was somewhat atypical of most white-rot fungi in that lignin degradation in laboratory tests was limited compared to cellulose degradation. Scurti (1956), however, grew *Armillaria* in vitro on pure cellulose and pure lignin, and observed that lignin was degraded but not cellulose. Whether these results reflect differences among species of *Armillaria* cannot be answered.

The ability to decay wood is probably quite different among species of *Armillaria*, and studies with known species are necessary. Marsh and Wargo (1989) found that some species of *Armillaria* produced high constitutive levels of an H_2O_2-enhanced phenol oxidase in vitro. This enzyme may be a lignin-degrading enzyme similar to the one found in the decay fungus *Phanerochaete chrysosporium* Burds (Tien and Kirk 1984).

This ability of *Armillaria* to decay wood after it has penetrated and killed the cambial tissues allows the fungus to maintain itself in woody tissues. Here it may build up inoculum potential and overcome the resistant reactions in the living cambial zone tissues, or infect and kill additional tissue when the tree is weakened by stress (fig. 3.9).

Host Factors

Physical barriers probably slow the penetration and infection of root tissue by *Armillaria*, but they do not prevent infection. Resistance is therefore mostly chemical as either preformed constituents in the bark or as mobilized constituents in response to penetration by the fungus. Limited work by Wargo (1984a) indicated that no increase in concentration of total or specific phenols occurred in bark tissues contiguous with bark naturally colonized by *Armillaria* or wounded and inoculated with the fungus. Since total bark was analyzed, the increase in phenols may have been masked. Other work indicates that phenol accumulation in bark tissue in response to fungal colonization occurs primarily in the inner bark regions (Ostrofsky and others 1984, Wargo 1988).

Preformed phenolics and other constituents can probably act as effective chemical barriers to penetration and infection by *Armillaria*. In vitro studies with *Armillaria* have shown that some phenols commonly found in both coniferous and deciduous hosts can inhibit fungus growth. Fifteen North American isolates representing at least the four species *A. mellea*, *A. gallica*, *A. ostoyae*, and *A. sinapina* (Wargo unpubl.) were challenged with hydrolyzable tannin (tannic acid, gallotannin) and gallic acid (Wargo 1980a). The isolates were both stimulated and inhibited depending on the phenol, the concentration of glucose, and the presence or absence of ethanol in the growth media (fig. 3.10). In general, gallic acid was more inhibitory to growth while hydrolyzable tannin was more stimulatory compared to the control. The ability to oxidize the phenolics seemed to be the key to inhibition or stimulation. Growth was inhibited if the isolate could not or only slightly (as determined by browning of the medium) oxidize the phenol. Growth was stimulated greatly where oxidation occurred readily; oxidation was initiated or accelerated by the addition of glucose and etha-

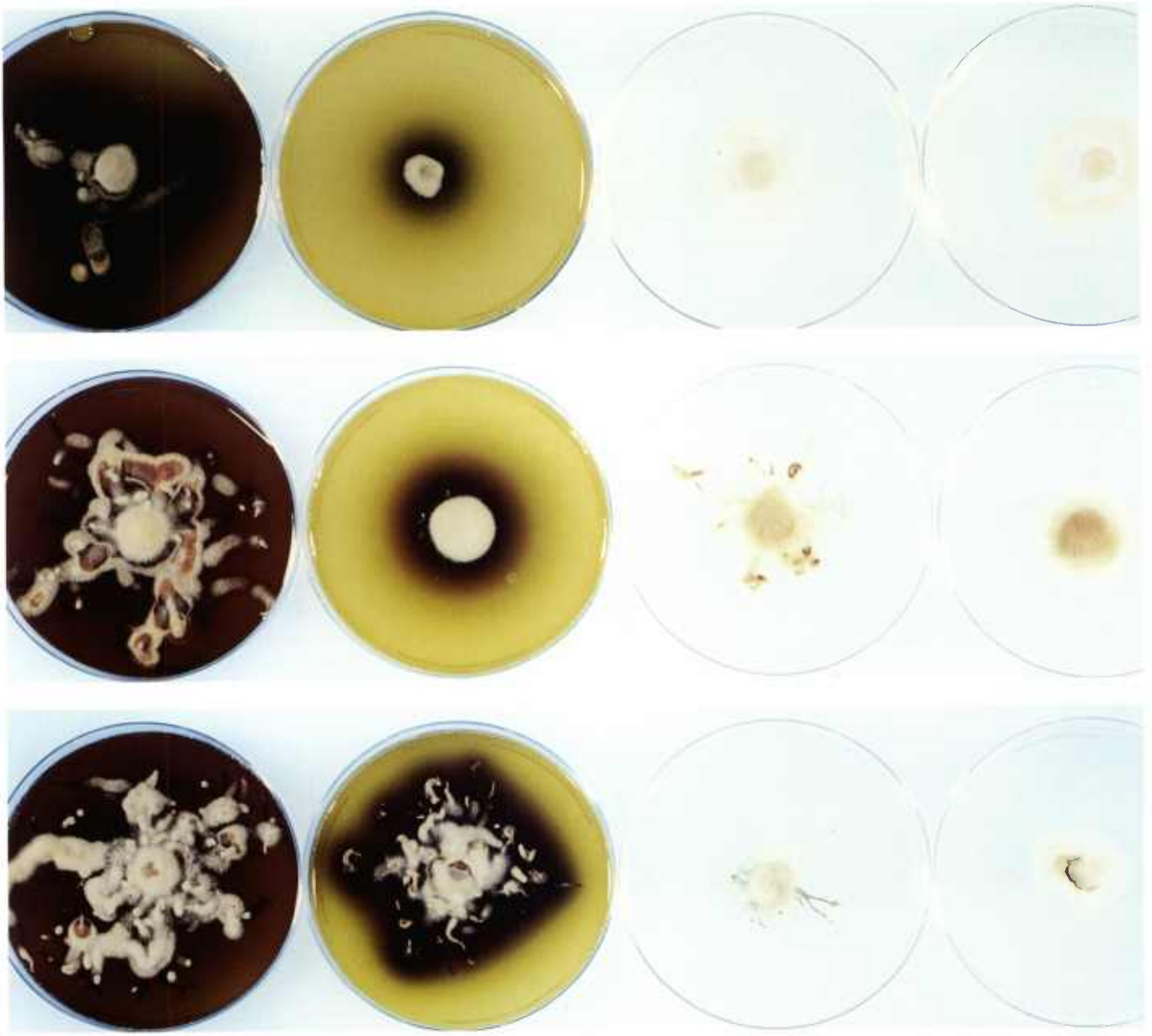


FIGURE 3.10 — Growth of an *Armillaria* isolate on gallic acid (GA) and control (C) media amended or not amended with ethanol (ET) and with three glucose levels (left to right, GA+ET, GA, C+ET, C). Top: 1 g glucose/l; Middle: 5 g glucose/l; Bottom: 10 g glucose/l. (P. Wargo)

nol. Isolates of *A. gallica* oxidized gallic acid and grew better in its presence with or without ethanol than did isolates of *A. ostoyae*.

Wargo (1981d) also observed that some ponderosa pine isolates of *A. ostoyae* from the Western United States that were pathogenic on the pine (Shaw 1977) could not oxidize gallic acid and did not grow at all on malt agar amended with 0.5% (w/v) gallic acid. Some less pathogenic western hardwood isolates (probably *A. gallica*, Shaw 1984) were able to oxidize gallic acid and responded similarly to eastern hardwood isolates that become pathogenic after stress has altered the tree (Wargo and Shaw 1985). Shaw (1985) could not confirm these reactions to gallic acid. He found that gallic acid both with and without ethanol inhibited most (20/21-dry weight, 21/21-colony diameter) of the 21 *Armillaria* isolates tested representing *A. mellea* (4), *A. ostoyae* (4), *A. gallica* (5), NABS V (3), *A. luteobubalina* (3), and *A. novae-zelandiae* (2). Variation within a species was as great as among the species. Growth of all isolates was stimulated on tannic acid medium (hydrolyzable gallo-tannin) without ethanol; with ethanol, a few isolates (4/21) grew less.

The different response of *A. ostoyae* isolates to gallic acid in both studies (Shaw 1985, Wargo 1981d) may have resulted from the different inocula used. Wargo

used inoculum growing on water agar and Shaw used inoculum from 3% malt agar. The isolates on malt agar may have been conditioned to produce laccase (malt agar turns brown when *Armillaria* isolates grow in it, indicating oxidase activity) and were able to oxidize some gallic acid immediately. Also, Shaw amended 3% malt agar with gallic acid while Wargo used 2% malt agar. The difference in nutrient concentration could have affected the abilities of the various isolates to oxidize gallic acid (Wargo 1980a). Cheo (1982) also observed a carbohydrate effect on *Armillaria* growing on tannin-supplemented media. Growth of a single isolate with 0.5% tannin was 1.5 to 5 times greater when glucose was added to the medium.

The stimulation of *Armillaria* species by tannic acid and the inhibition by gallic acid suggests that the concentration of gallic acid and the rate at which it can be oxidized controls the response of the fungus. Tannic acid has approximately one glucose molecule for every five gallic acid molecules. The fungus may hydrolyze tannic acid to gallic acid, which it then oxidizes and polymerizes immediately. This prevents the gallic acid concentration from becoming inhibitory. Alternatively, the fungus may oxidize the tannin without hydrolyzing it, thus preventing gallic acid from building up in the substrate. No work has been conducted on degradation of tannins by *Armillaria*. Analyses of phenols and tannin degradation in oak bark tissues colonized by *Armillaria* showed that gallic acid did not occur in colonized tissue (Wargo 1984a). Gallic acid and various polymers (di, tri, etc.) of gallic acid were present in the healthy and discolored tissues contiguous with the colonized portion but these materials decreased in the colonized bark compared to healthy tissues. This suggests that *Armillaria* oxidizes tannic acid and other polymers of gallic acid but does not hydrolyze them to gallic acid. However, this needs to be verified with more critical experiments.

The ability of *Armillaria* to oxidize gallic acid, tannic acid, and other phenols in bark tissues is also influenced by carbon and nitrogen concentrations (Wargo 1983b). The growth rate and hence oxidation rate of phenols in extracts from root bark of black oak depended on supplemental glucose and nitrogen. Growth was directly proportional to the decrease in level of total phenols in a culture medium, and was five times greater in the phenol plus supplement medium than in supplement alone.

Phenols other than gallic acid and gallotannins also can inhibit *Armillaria* species. Both *A. ostoyae* and *A. gallica* were inhibited by various monophenols and alpha pinene, a terpene in conifer resins (Entry and Cromack 1989). Low levels of these phenols (<1 mg g^{-1}) stimulated rhizomorph production. No differences occurred between the two *Armillaria* species in response to the various phenols or pinene; variation of growth response to each compound was as great within as between species. These results must be accepted very cautiously because the compounds were dissolved in 50 ml ethanol and added to 1 l of medium. This concentration of ethanol is 30 to 100 times greater than concentrations used in other studies. Results could be confounded by these high concentrations. Alkaloids are also known to inhibit *Armillaria*. Greathouse and Rigler (1940) found that alkaloids from several plant families inhibited growth of *Armillaria* in vitro.

Other plant constituents have been found highly stimulatory to *Armillaria*. Lipids from roots of ponderosa pine, Douglas-fir, white fir, incense-cedar, and peach promoted vigorous growth in vitro of an *Armillaria* isolate from California, probably *A. mellea* (Moody and Weinhold 1972a,b). The fatty acid fraction of the lipids was the active portion. Resin acids from ponderosa pine also were highly stimulatory and promoted twice as much rhizomorph growth as the fatty acid fraction from the same amount of root tissue. Abietic acid, a commercially available resin acid, stimulated rhizomorph production when it was sterilized by autoclaving but not by filtration, suggesting that breakdown products of the acids are the stimulatory factors. Fresh or autoclaved wound resin from ponderosa pine also stimulates in vitro growth of *Armillaria* (Shaw 1975) and has been used in medium prepared for cultural paring tests (Shaw and Roth 1976).

Predisposition Effects

Stress

Susceptible or resistant responses of the host to a fungal pathogen depend on the genetic makeup of the host and the pathogen, and the environment in which they exist. Stress can alter the relationship and change the balance in the interaction between host and pathogen, resulting in root disease.

Stresses obviously affect the pathogen, but few studies report on these effects. We know that drought and waterlogging sometimes increase the incidence and severity of *Armillaria* root disease (see chapter 7). However, we have no idea how drought or waterlogging affect the fungus when it occurs as rhizomorphs in the soil or as mycelium inside tree tissues. For example, we do not know how turgor pressure in the rhizomorph influences penetration of the root bark; nor do we know how moisture extremes influence this relationship. Nechleba (1915), in his conclusions regarding the pathogenic relationship of trees and *Armillaria*, specu-

lated that dry conditions in forests promoted infection and colonization by inducing rhizomorphs of the fungus to colonize other substrates for water and nutrients. He proposed that the rhizomorphs "find their way instinctively (hydrotropism) toward living roots" and colonize them.

Armillaria species infect roots of healthy trees by rhizomorph contact, from diseased tissue, or by direct mycelial contact from diseased roots (see chapters 4 and 6). Hyphae penetrate the outer bark and "challenge" the inner bark tissue; it is here where stress influences the reaction. Chemical changes induced in the host by stress may promote susceptibility by (1) removing fungal inhibitors, (2) releasing nutrients and metabolites required by the fungus for pathogenesis, (3) providing the fungus with growth stimulators that allow it to overwhelm the capacity of the host root system to resist harmful fungal metabolites, or (4) reducing the capacity of the host tissues to tolerate or control the metabolites produced by the fungus (Wargo 1984b). All or any combination of these relationships may occur.

Many stresses predispose trees to *Armillaria* and initiate root disease or accelerate root disease in the host (see chapter 7). However, our knowledge about how stress specifically affects the relationship between *Armillaria* and its hosts is mostly about the host and is limited predominantly to the effects of drought and insect damage on a few host tree species (Wargo 1983a,b; 1984a,b).

Nutritional Changes

Both drought and defoliation affect the carbohydrate and nitrogen levels in the root tissues colonized by *Armillaria* (Gregory and Wargo 1986, Parker 1979, Parker and Houston 1971, Parker and Patton 1975, Wargo 1972, Wargo and others 1972). Defoliation can substantially decrease the starch content in the root wood (fig. 3.11) and decrease sucrose levels in both bark and cambial tissues of sugar maple roots (Wargo 1972, 1981b). Reducing-sugar levels increase especially in cambial zone tissues. Concentrations of reducing-sugar can be 4-5 times higher in defoliated trees than those in non-defoliated trees at the same time of the year, and 3-4 times higher than the normal spring high when carbohydrates are mobilized for growth (Wargo 1971). Since *Armillaria* predominantly uses glucose (Garraway 1975, Wargo 1981a), this increase is potentially important to the fungus. Growth on glucose or polymers of glucose, such as maltose and starch (fig. 3.12), can be 1.5-3 times higher than growth on other carbon sources (Wargo 1981a). Enhanced growth of *A. calvescens* (Wargo unpubl.) on root extracts of defoliated sugar maples was related to higher levels of glucose in the extract (Wargo 1972).

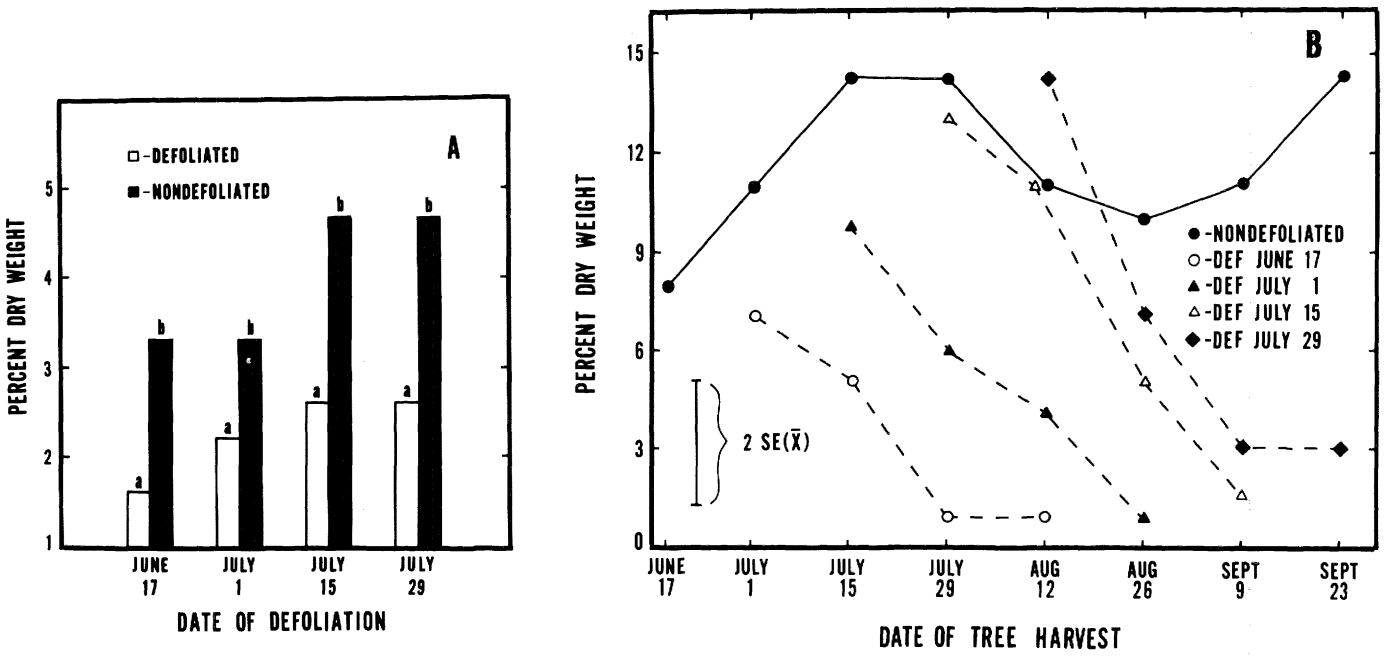


FIGURE 3.11 — Decline in sucrose and starch content in the bark and wood of sugar maple roots caused by defoliation. A: Sucrose level in the inner bark; B: Starch level in wood. (From Wargo 1972)

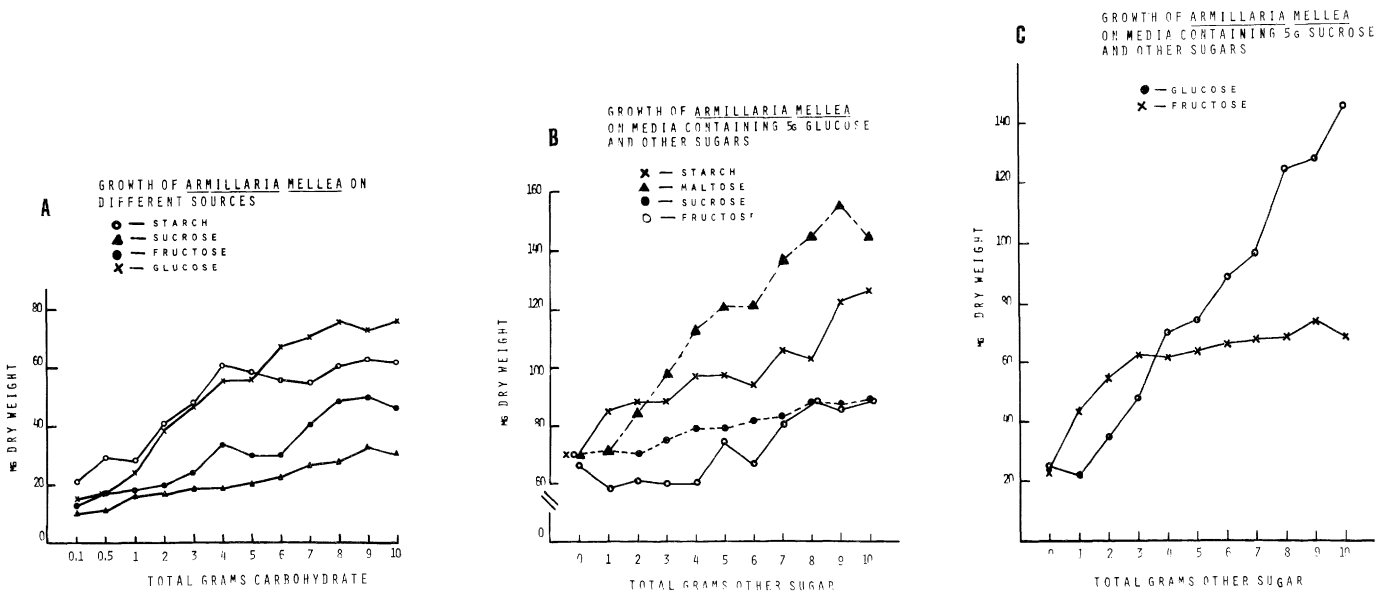


FIGURE 3.12 — Growth in vitro of *Armillaria* on various carbohydrates that demonstrate the stimulation of growth by glucose. A: Growth on various carbohydrates; B: Growth on glucose media supplemented with various carbohydrates; C: Growth on sucrose media supplemented with glucose and fructose. (From Wargo 1981a)

Drought and defoliation also increase both total amino nitrogen levels and certain individual amino acids in sugar maple trees (Wargo 1972) and seedlings of black and red oak (Parker 1979, Parker and Patton 1975). Both individual amino acids and total animo nitrogen supplements were very satisfactory nitrogen sources for in vitro growth of *Armillaria* (Weinhold and Garraway 1966), as discussed previously.

Also, noted earlier, ethanol is a potent stimulator of *Armillaria*, especially rhizomorph production and growth (Weinhold 1963, Weinhold and Garraway 1966). In the presence of ethanol, the fungus can metabolize phenolic compounds that would otherwise inhibit growth (Longworth and Garraway 1981; Wargo 1980a, 1981d). Ethanol enhances laccase production by the fungus (Worrall and others 1986) and improves its ability to utilize carbon sources other than glucose (Weinhold and Garraway 1966).

Ethanol could be an important factor in stressed trees. Stress from flooding or defoliation can stimulate ethanol production and accumulation in woody roots (Wargo unpubl.). On poorly drained sites and more mesic areas, seasonally high water tables often occur and cause anaerobic conditions about tree roots. Defoliation, because it reduces transpiration, promotes or prolongs wet soil conditions. In oak forests in Connecticut, soils in stands defoliated by the gypsy moth (*Lymantria dispar* L.) were wetter and defoliated trees contained more water than soils and trees on adjacent nondefoliated sites (Stephens and others 1972). Significant amounts of ethanol can be produced in roots depending on the duration of the anaerobic conditions and tree species (Coutts and Armstrong 1976, Crawford and Baines 1977). Injection of ethanol into roots of black and white oaks promoted colonization of the roots by *Armillaria*. Colonization, however, was related more to tissue necrosis caused by the ethanol rather than to the ethanol alone (Wargo and Montgomery 1983).

Phenol Degradation

Stress-induced chemical changes in roots may also determine how well *Armillaria* can oxidize phenols. Inhibition of *Armillaria* growth by gallic acid was lessened or reversed by adding more glucose to the medium (Wargo 1980a). Growth in bark extracts from black oak roots depended on phenol oxidation, which was greatly enhanced by adding glucose and nitrogen to the extract (Wargo 1983b). Additional growth studies using commercial sources of phenols found in oak bark (quercetin, quercitrin, catechin, and tannic acid) indi-

FIGURE 3.13 — Growth of an *Armillaria* isolate on an extract from red oak bark. Upper flask—extract + glucose + ethanol. Lower flask—as above + 500 ppm ascorbic acid. (P. Wargo)

cated that if the fungus could oxidize the phenol, the phenol no longer inhibited the fungus (Wargo unpubl.). Growth was also stimulated, suggesting that the oxidized phenols were being utilized as carbon sources or growth regulators. If oxidation of the phenols were inhibited by adding a reducing agent (fig. 3.13), growth significantly declined (Wargo unpubl.).

Successful colonization of root tissues in stressed trees may depend on the fungus' ability to oxidize phenols and the inability of the tree tissue to prevent the oxidation reaction. In healthy deciduous trees, *Armillaria* appears to be confined to wounded and necrotic tissue; contiguous healthy tissues are not "browned" or colonized by the fungus. In weakened trees, contiguous living tissues are "browned" in advance of the fungus, probably by extracellular secretions of laccase and peroxidase, and then colonized (Wargo 1983b, 1984a). This interaction has similarities to that proposed for the redox theory of hypersensitivity reaction (Goodman and others 1986) where necrosis in response to fungal invasion occurs when the balance between reductive and oxidative processes shift in favor of the latter. In healthy tissues, necrosis induced by *Armillaria* is inhibited or contained, probably by a highly reductive state in contiguous tissues. Perhaps stressed tissues cannot confine the oxidative processes and necrosis begins and spreads as oxidative and other enzymes are secreted by the fungus.

Host-Induced Lysis

Host-produced enzymes that may potentially assist bark tissue in resisting *Armillaria* are also affected by stress from defoliation (Wargo 1976). The hyphal walls of *Armillaria* contain chitin and beta-1,3-glucan, and are vulnerable to lysis by chitinase and beta-1,3-glucanase (Ballesta and Alexander 1972, Bouveng and others 1967, Wargo 1975). These enzymes are found in bark and sap of several oaks and sugar maples, and their activities are lowered by defoliation (Wargo 1975, 1976). Lysis of *Armillaria* hyphae in vivo has been reported for species associated with orchids (Hamada 1940, Kusano 1911) and the description of fungal digestion in orchid species suggests a host-mediated lysis (Burges 1939).

Complete dissolution of the hyphae is not necessary to disrupt growth. Hyphal tips grow by a delicate balance between wall synthesis and wall lysis, and bursting of the hyphal tips can occur when the balance shifts toward the lytic stage (Bartnicki-Garcia and Lippman 1972). Extrahyphal enzymes in host cells that can dissolve hyphal wall components could alter the wall formation balance, disrupt hyphal-tip growth, and provide a defense mechanism against invasion by fungal pathogens. More recent work on these enzymes indicates that they are indeed potent inhibitors of fungal growth (Schlumbaum and others 1986).

The fungus is not defenseless against lysis by host-produced enzymes. The phenol oxidase enzymes, especially tyrosinase, produced by the fungus are linked to melanin synthesis by fungi (Mayer and Harel 1979). As noted earlier, *Armillaria* is capable of producing melanin-like pigments in rhizomorphs and probably to a limited extent in hyphae (Chet and Hüttermann 1977, Smith and Griffin 1971). Phenol oxidase-catalyzed formation of extracellular pigments may be related to the formation of melanin-like pigments in hyphae. They may strengthen hyphae (Bell and Wheeler 1986) and protect them from dissolution by lytic enzymes (Bloomfield and Alexander 1967).

Conclusions

Host-pathogen interactions ultimately depend on the relationship of fungal species, host species, and the environment in which they interact, including the disturbances induced by stress. Much of the information on the physiological and chemical interactions of *Armillaria* species and their hosts is fragmented, and the characteristics of the events for any one species of

Armillaria and its host are incomplete. The fungus penetrates generally through intact bark, interacts with the inner bark, is stimulated to colonize and kill the inner bark, and either invades the cambial zone or is inhibited by as yet unknown mechanisms. The interaction with phenols present in the bark tissues is probably a major event in determining resistance or susceptibility and the pathogenic process. Stress from a variety of sources influences the resistance mechanisms and enhances penetration, colonization, and killing by *Armillaria*.

The concepts discussed in this section are based on fragments of information concerning the many interactions that can occur among the many *Armillaria* species and host species. Studies using clonal host material, known species, and genotypes of *Armillaria* and stressed and non-stressed systems must be conducted to elucidate the kinds and sequence of pathogen and host changes that occur in resistant and susceptible reactions. Some of the morphological and anatomical interactions have been characterized. These must be verified in the host-pathogen system described above, and the chemical changes associated with these interactions must be characterized. This area of research is ripe for much work by the students of host and fungal physiology and their interactions.

CHAPTER 4

Inoculum and Infection

Derek B. Redfern and Gregory M. Filip

All *Armillaria* species survive saprophytically in woody substrates in soil, and the majority form the most highly organized rhizomorphs of any fungus. By extension of these rhizomorphs through the soil, the fungus can colonize additional woody material. Varying degrees of pathogenicity may be exhibited during this phase. Robert Hartig (1873b) was the first to not only make the link between the spread of infection and the presence of nearby trees previously killed by the fungus, but also to suggest that rhizomorphs cause infection.

Descriptive terms such as "food base" and "invasive potential" have obvious application to the rhizomorph-forming *Armillaria* species. "Inoculum potential" is a similar term. This concept was explored by Garrett (1970), partly through a series of experiments with *A. mellea (sensu lato)* (Garrett 1956b). The term was not new, but he redefined it (1970) as "the energy of growth of a parasite available for infection of a host, at the surface of the host organ to be infected." The definition encompasses the net effect of variables such as the surface area of fungus in contact with unit area of host, the vigor of the invading hyphae, and environmental effects on the fungus.

This chapter deals primarily with factors that affect the success of infection through their effect on inoculum potential. First, the nature of the inoculum capable of causing infection and the quality of the substrate provided by different tree species are considered. The second part concentrates on those factors which affect the success of infection through their effect on the fungus, particularly the rhizomorphs, which provide the means of infection and spread in most *Armillaria* species.

Inoculum

Source of Inoculum

For all practical purposes, wood provides the only effective substrate from which *Armillaria* can spread and cause infection. Tree roots constitute the major source of inoculum, but logging debris may also be colonized and act in the same way (MacKenzie and Shaw 1977).

The fungus becomes established in roots and stumps by infecting live trees and by colonizing stumps created during felling operations. If a tree is killed, the entire root system may become inoculum. The fungus colonizes newly created stumps in three ways: by rapid extension from pre-existing lesions in which it was formerly held in check by host resistance (Kile 1980b, Leach 1939); by invasion from an epiphytic position on the roots; or by invasion from outside by newly arrived rhizomorphs.

Based on Garrett's work (1960, 1970), the series of circumstances under which *Armillaria* becomes established in substrates can be taken to represent a requirement for a decreasing parasitic ability and an increasing competitive saprophytic ability. Logging residues constitute an extension to the series because, apart from being less readily available for colonization than stumps by virtue of position, their tissues are likely to die more rapidly and be available earlier for colonization by competing saprophytic organisms.

Where stumps provide potential sources of inoculum, they are most commonly colonized by vegetative spread, but the cut surface can also provide an avenue for colonization by basidiospores (Rishbeth 1970, 1978b, 1988). A number of researchers have failed to infect stumps in this way, however (Kile 1983b, Leach 1939, Podger and others 1978), while others have had very limited success (Swift 1972). It is apparently an uncommon event but may be important to disease development in certain crops (Horner 1988). Even though basidiospore-infected stumps probably constitute a minor portion of the total inoculum, spore infection is important for providing a source of genetic diversity, for facilitating long-range spread, and also for infecting forests established on arable land. Some work on genotype identification (Hood and Sandberg 1987, Horner

1988, Kile 1983b, Ullrich and Anderson 1978) provides indirect evidence for spore infections, but similar work by others provides less support (Shaw and Roth 1976).

No evidence indicates basidiospores can directly infect living roots, presumably because the inoculum potential provided by the limited resource within spores is inadequate. Hartig (1874) suggested that basidiospores may colonize dead organic matter and subsequently form rhizomorphs, but no experimental evidence supports this.

In experiments, most successful infections have been achieved using woody inocula prepared either from naturally infected roots (Leach 1937) or by culturing the fungus in various ways on woody stem or root segments (Patton and Riker 1959, Redfern 1975, Shaw 1977, Thomas 1934). Cultures established on non-woody substrates such as nutrient agar, bran, or bean pods have been generally unsuccessful as inocula (Bliss 1941, Plakidas 1941). Wood is not an absolute prerequisite for infection; inocula derived from less substantial substrates may be adequate. For example, Guyot (1927) caused infection using cultures on an agar medium containing acorns and horse chestnuts. Nevertheless, only a woody substrate is able to provide an inoculum which is sufficiently durable and potent to cause disease reliably.

Under experimental conditions, infection has been achieved even without a substrate by means of excised rhizomorphs. These pieces can be large enough to form new growing tips with an inoculum potential high enough to infect healthy seedlings (Redfern 1973, Rykowski 1984). Holdenreider (1987) caused infection in a similar way but found wounds to be an apparent prerequisite. Other reports concerning the infective potential of detached rhizomorphs have been negative (Bliss 1941).

In common with other root-rot fungi, *Armillaria* inoculum is generally confined to infested sites. However, roots may become fragmented and transported by water, thus potentially creating new foci of infection (Hewitt 1936). Colonized logging debris could be transported in the same way. The rhizomorph-forming ability of most species would enable *Armillaria* to exploit such an event much more effectively than other root pathogens such as *Heterobasidion annosum* (Fr.) Bref. and *Phellinus weirii* (Murr.) Gilbn.

Substrate Quality—Conifers Versus Hardwoods

Armillaria mellea sensu lato was considered to be a highly variable species long before the present understanding of speciation in the genus and of the ecology of these species. In spite of this, much of the observed variation in disease was attributed to factors other than variation in pathogenicity. Prominent among these was the nature of the substrate providing the inoculum.

Disease is now known to be associated with stumps of many species, ranging from Australasian hardwoods (Kile 1981, Podger and others 1978, Shaw and Calderon 1977) to European and North American conifers (Redfern 1975, Shaw and others 1976a). Early records, however, largely associated mortality with hardwood stumps. A possible reason for this is that until relatively recently the disease attracted most attention in fruit orchards and in plantations of tea, coffee, rubber, and exotic conifers, all established on land cleared of indigenous forest where *Armillaria* was endemic. In the tropics and sub-tropics, this original forest comprised a mixture of broadleaved species (Leach 1939). In temperate regions, hardwoods would probably have been at least a major component on the richer soils where such plantation crops were grown. Many early reports of disease concern losses in these circumstances (Butler 1928, Dade 1927, Gibson 1960, Hendrickson 1925, Horne 1914, Lawrence 1910, Nechleba 1915, Rhoads 1956, Wallace 1935). In California, the disease occurred so consistently in orchards planted on land cleared of oaks that for many years articles in Californian agricultural journals referred to *Armillaria* as the "oak root fungus" (Kimball 1949, Raabe and others 1967).

In Europe, Hartig (1874) and Nechleba (1915) observed that serious disease may occur where conifer plantations replace hardwoods, whereas damage is generally unimportant in crops replacing conifers. This had a major influence on early thinking about how substrate affects disease development. The prevailing view was that hardwood stumps provide a superior substrate to conifer stumps. Peace (1962), for example, commented that *Armillaria* is essentially a fungus of areas with a hardwood history, and suggested that where conifers replace hardwoods damage is likely to be absent or much reduced in the second conifer rotation. During the first rotation, conifer stumps left after thinning are readily colonized by *Armillaria* (Greig 1962, Low and Gladman 1962), but Peace (1962) believed the fungus acts purely saprophytically in this situation and there is no increase in parasitic activity. The implication was that conifer stumps have little or no significance in sustaining attacks.

By contrast with observations implicating hardwood stumps as sources of infection, the first reports in which disease was clearly identified as being associated with conifer stumps are relatively recent. Weiss and Riffle (1971) recorded killing of ponderosa pine following a crop of the same species, and Swift (1972) reported losses in slash pine planted as a second rotation

on a site formerly occupied by indigenous hardwoods. Ono (1965, 1970) and Redfern (1975) reported serious disease where the major source of nutrition for the fungus was provided by conifer stumps. Initially, such observations were rare among the continuing reports concerning hardwoods (Gladman and Low 1963, Huntly and others 1961, MacKenzie and Shaw 1977, Ono 1965, Pronos and Patton 1978, Swift 1972). They have become more numerous, particularly from natural coniferous forests in North America (Morrison 1981, Wargo and Shaw 1985), as increasing interest in forest management draws attention to the impact of *Armillaria* losses. In the Northwestern United States, conifer stumps were shown to be effective inoculum sources (Filip 1979, Roth and others 1980) causing considerable infection and mortality in several indigenous coniferous species, especially in partially harvested forests (Filip 1977, Filip and Goheen 1984, Shaw and others 1976a).

In experiments, trees have been successfully infected using inocula prepared from stems and roots of various coniferous and hardwood species, providing ample evidence of at least the short-term suitability of coniferous substrates as food bases for *Armillaria*. Species used include red pine and eastern white pine (Patton and Riker 1959); Japanese larch (Ono 1970); fig and citrus (Wilbur and others 1972); common beech, planetree and Scots pine (Redfern 1975, 1978); Sitka spruce (Singh 1980a); alder (Shaw 1977, Shaw and others 1981); and English oak (Morrison 1982b).

While rhizomorph production may not be the best measure of substrate quality, particularly for those pathogenic species which produce few rhizomorphs, it has been commonly used. Thus, in experiments to determine the relative value of the substrate provided by roots of hardwood and coniferous species, Redfern (1970) found that segments of red maple inoculated with *Armillaria* produced a greater number, total length, and dry weight of rhizomorphs than red spruce segments of equal volume. However, when corrections were made for differences in initial wood density of the two species, differences in length and weight were no longer evident, although maple segments still produced a greater number of rhizomorphs than spruce. Working with several *Armillaria* isolates and several coniferous and hardwood species as substrates, Morrison (1972) found that, with the exception of one isolate, hardwood segments produced a greater dry weight of rhizomorphs than conifer segments. He made a similar correction for density. The number of rhizomorphs was not assessed in this experiment, but when stumps were inoculated in the field, Morrison found that the number of rhizomorph systems, as well as the total length of rhizomorphs per stump, was greater for hardwood stumps than for conifer stumps. In a similar study, which included measuring rhizomorph production by naturally infected stumps, Rishbeth (1972b) concluded that pines are inferior to English oak as substrates for *Armillaria* in terms of the number and weight of rhizomorphs produced. In comparing maritime pine with oak, Guillaumin and Lung (1985) obtained the same results as Rishbeth for both *A. ostoyae* and *A. mellea*.

Redfern (1975) examined the effect of substrate on infection as well as rhizomorph production. Sitka spruce seedlings were inoculated with four isolates of *Armillaria* growing on root segments of either planetree or Scots pine. Gregory (1985) subsequently identified these isolates to species. *Armillaria ostoyae* and *A. mellea* killed more trees when growing on planetree than on pine, whereas the reverse was true for *A. gallica*. Substrate species had no effect on *A. cepistipes*. Rhizomorph production was significantly greater on planetree than on pine for three of the species (*A. ostoyae, A. gallica,* and *A. mellea*), but *A. cepistipes* produced more on pine. *Armillaria ostoyae* and *A. mellea* were both highly pathogenic in the experiment, whereas the other two species showed very low pathogenicity. Thus, for both pathogenic species, rhizomorph production and infection were favored by a hardwood rather than a coniferous substrate. Rykowski (1984) obtained similar results in experiments with Scots pine seedlings and inocula prepared from branch segments. Hardwood substrates, especially oak and common beech, were superior to Scots pine and European larch for both rhizomorph production and infection. Three isolates were used, but only one produced rhizomorphs consistently and caused infection. The species was referred to as *A. mellea,* but evidence in the paper suggests it was *A. ostoyae.*

In similar work with the Australasian species *A. novae-zelandiae* and *A. limonea,* Benjamin and Newhook (1984b) ranked a number of indigenous and exotic hardwood species and two exotic conifers, radiata pine and ponderosa pine, as substrates for rhizomorph production. The two conifers occupied an intermediate position among the hardwoods as food bases for *A. novae-zelandiae,* whereas they were equal or superior to most of the hardwoods for *A. limonea*. Interestingly, the native hardwood tawa provided the best substrate for both species. In pathogenicity trials using the two *Armillaria* species with radiata pine and eucalypt seedlings, radiata pine and several hardwood food bases were equally effective substrates when tested against radiata pine seedlings. Some evidence indicated that tawa was superior to radiata pine against eucalypt seedlings.

Pearce and Malajczuk (1990a) tested the quality of the food base provided by two common hardwood hosts of *A. luteobubalina* by measuring rhizomorph production.

They found that stem segments of sunbush were superior to those of karri. Three genotypes of *A. luteobubalina* behaved in the same way.

With so few experiments on substrate quality, data are insufficient to suggest a general superiority of one wood type over the other as a food base, but some *Armillaria* species or isolates may be favored by particular species. However, observations similar to those made by Nechleba (1915) concerning the association of killing attacks with former hardwood sites continue to be made (Rishbeth 1982, Rykowski 1984). In the field, factors other than the intrinsic quality of the substrate may determine a stump's effectiveness as an inoculum source. Morrison (1972) and Rishbeth (1972b) both concluded that the frequently reported association of hardwood food bases with disease could be partially attributed to those broadleaved trees in which resistance to infection is maintained by regrowth after cutting. They are less quickly exhausted as food bases than conifer stumps, which die rapidly. The generally higher wood density and greater resistance to decay of hardwood species compared to conifers (Rykowski 1984) may also increase the longevity of hardwood inocula.

The possible "field" superiority of hardwood food bases as inoculum, at least for some *Armillaria* species, is not great, and the association of disease with hardwood stumps should not be over-emphasised. As discussed by Redfern (1975), it may be a mistake to assume that damage will diminish appreciably in succeeding conifer rotations. This is supported by recent survey data from second-rotation radiata pine stands established on land cleared of indigenous hardwood forest (MacKenzie and Self 1988). It is salutory to quote Hartig, who wrote in 1874: "The disease often occurs especially destructively where the planting of conifers has been carried out after the felling of hardwoods .... But it should not be maintained from this that the rhizomorphs attack only from hardwood stumps to the conifer woods since, as we said earlier, the mycelium grows for several years on all conifer stumps and roots; therefore, hardwood stumps are not necessary for the spread or origin of the disease."

The nature of the substrate probably has far less direct influence on disease development in plantations than the pathogenicity of the *Armillaria* species present in the previous crops. However, an indirect substrate effect may occur through species selection resulting from host specialization. Thus, Rishbeth (1985a) found that despite being rare on broadleaved trees and stumps, *A. ostoyae* caused death as commonly as *A. mellea* in conifers established on sites previously occupied by broadleaved woodland. Where conifers replaced conifers, it was the predominant cause of mortality.

The importance of variation in pathogenicity between species is suggested in the early North American literature. In a notable paper, Piper and Fletcher (1903) described damage in prune orchards by two forms of *A. mellea (sensu lato)*. One form, referred to as *A. mellea*, caused severe damage and was believed to have been introduced. The other, referred to as *A. mellea bulbosa*, was much less damaging. The latter was abundant on native trees, both conifers and hardwoods. Later, Childs and Zeller (1929) observed disease in apple orchards established on sites cleared of oak, but found no disease on sites formerly occupied by Douglas-fir. Both site types were infested with *Armillaria*, which the authors suggested might exist as two strains differing in "pathogenicity" (see chapter 6).

Substrate Specialization

In common with other wood-rotting fungi that kill tree roots, *Armillaria* is polyphagous. Individual species or isolates grow on excised stems or roots of many tree species, including ones which they would not encounter naturally (Benjamin and Newhook 1984b, Rishbeth 1978a). There is little prima facie evidence for substrate specialization. In the field, however, substrates are acquired both parasitically and saprophytically. Where several *Armillaria* species of different pathogenicity and competitive saprophytic ability are present in the same forest type, substrates are unlikely to be equally available to them all. Our knowledge of the better-known species clearly shows that their association with particular substrates reflects their ecology rather than a substrate specialization or preference.

Armillaria ostoyae is highly pathogenic and occurs mainly on conifers throughout Europe and North America (see chapters 6 and 8). However, its association with conifers is not exclusive. In Canada, Morrison and others (1985a) found that broadleaved trees within disease centers were frequently attacked and killed. Elsewhere in North America, *A. ostoyae* kills cherry (Proffer and others 1987) and several other hardwood species (Harrington and others 1989). By contrast, Europe's other major pathogenic species, *A. mellea*, may be described as a "hardwood species" because it has a wide host range among hardwood trees and shrubs, and is common on hardwood stumps (Guillaumin and others 1985, Rishbeth 1985a). The association is not exclusive, however, as it also attacks young or weakened conifers and occasionally occurs on conifer stumps (Davidson and Rishbeth 1988, Rishbeth 1985a). *Armillaria gallica* also has a wide host range (Guillaumin and others 1985) and has been recorded as a weak pathogen on both hardwood and coniferous hosts, but it is most important as a cause of butt rot in hardwood trees and as a colonist of hardwood rather than conifer stumps (Rishbeth 1985a). Morrison and

others (1985a) found *A. gallica* exclusively on living and dead hardwood hosts.

Experiments show that whereas only *A. mellea* and *A. ostoyae* infect vigorous English oak and Scots pine, respectively, all three species colonize both hosts when resistance is reduced by suppression (Davidson and Rishbeth 1988). Neither host specialization by the fungus nor selectivity by the tree are apparently maintained under these circumstances. Thus, for *A. ostoyae* and *A. mellea*, their host specialization as primary parasites largely determines their substrates as saprophytes.

Kile and Watling (1983, 1988) have discussed the ecology of the five known Australian species (see chapter 8). Four of these species, *A. luteobubalina, A. hinnulea, A. novae-zelandiae,* and *A. fumosa,* have extended geographical distributions which include Tasmania. *Armillaria hinnulea* and *A. novae-zelandiae* also occur in New Zealand. Some species overlap ecologically, but the last two species occur in wet forests, whereas *A. luteobubalina* predominates in dry sclerophyll eucalypt forests. *Armillaria fumosa* has only been found on wet sites within these dry forests, and is therefore associated with the particular species of these locations.

Armillaria luteobubalina is the only Australasian species for which comprehensive information about substrate species is available, but it does not indicate substrate specialization among the hosts commonly present in the dry sclerophyll eucalypt forest. Both stumps and trees of the major eucalypt species groups are equally susceptible to infection (Kellas and others 1987, Pearce and others 1986, Shearer and Tippett 1988); its host range includes 81 species in 21 plant families (see table 8.1).

In New Zealand, *A. limonea* and *A. novae-zelandiae* cause serious disease in radiata pine established on sites formerly occupied by indigenous forest comprising host species such as tawa and rimu (MacKenzie and Shaw 1977). However, no evidence indicates that certain species provide superior substrates or that they are preferred substrates for one *Armillaria* species or the other (MacKenzie and Shaw 1977, van der Pas 1981a).

In general, there is little evidence for substrate specialization within the natural range of each *Armillaria* species. The New Zealand example provides a dramatic illustration since the two species involved appear to have transferred successfully from indigenous hardwoods to a northern-hemisphere conifer (MacKenzie and Self 1988). Nevertheless, in northern temperate forest types, several species express a degree of specialization since *A. mellea* and *A. gallica* are generally associated with broadleaved hosts and *A. ostoyae* with conifers.

The infection of stumps by basidiospores offers, in a sense, a "free choice" of substrate. Rishbeth (1988) made the interesting observation that *A. ostoyae* and *A. gallica* most frequently colonized conifer and hardwood stumps, respectively, although both species also colonized the other substrate.

Longevity of Inoculum and Persistence of the Fungus

Most estimates of inoculum longevity are based on observations made on single occasions, and refer to the ages of stumps which show evidence of viable *Armillaria*. Observations of this nature offer no information on the persistence of the fungus on the site and may underestimate its longevity in individual stumps. For example, survival in the stumps of hardwood trees showing regrowth may be greatly affected by the extended period over which such stumps become colonized. When the fungus is already present as a perthophyte or as a butt rot, colonization may begin long before the tree is felled. Thus, longevity of the fungus in individual roots may give little idea of the time over which the stump may act as an inoculum source.

Estimates vary widely but generally indicate fungal survival for decades in both broadleaved and coniferous stumps. Pronos and Patton (1978) found that oaks killed by herbicide produced rhizomorphs for at least 14 years, and Rishbeth (1972b) reported that wood from English oak stumps could do so 40 years after the trees were cut. Swift (1972) gave a figure of at least 20 years for survival in East African hardwoods. The only data available for conifers are from ponderosa pine in North America, and probably refer to *A. ostoyae.* Shaw (1975) found that wood cut from 30-year-old stumps contained viable *Armillaria* which could produce rhizomorphs; Roth and others (1980) isolated the fungus from large, old-growth stumps more than 35 years old. They estimated that it would remain viable in such stumps for at least 50 years. Few data are available for identified species. Kile (1981) suggested a longevity of 15-25 years for *A. luteobubalina* in messmate stringybark. In contrast, he isolated *A. hinnulea* from 70-year-old stumps of the same eucalypt species (Kile 1980b and pers. comm.). Rishbeth (1985a) recently reported an example in which *A. gallica* remained viable in an oak stump 53 years after felling.

Making valid comparisons between species based on field observations is difficult since longevity is likely to be affected by the stump species, its size, and by environmental factors. The difference in longevity between *A. luteobubalina* and *A. hinnulea* quoted above might be attributable largely to site differences since the observations were made in different forest types (G.A. Kile,

pers. comm.). There are some indications from experiments with small inocula about the effects on survival of soil moisture (Pearce and Malajczuk 1990a), temperature (Bliss 1946), and competing fungi such as *Trichoderma viride* Pers.: Fr. (Garrett 1957) but further work is required. Inoculum size may not be a major factor. Even in the comparatively minute inocula used in experiments, the fungus remained viable in Sitka spruce for at least 4 years (Singh 1980a) and in pine for up to 3 years (Patton and Riker 1959).

Armillaria can persist on a site for a very long time. For example, Shaw and Roth (1976) suggest that individual clones of *A. ostoyae* may survive for several centuries. Clearly this must involve a succession of substrates. For pathogenic species, these may be acquired either at the margins of expanding disease centers or among regenerating trees within disease gaps following a period of survival in stumps. The figures cited for longevity in individual stumps suggest this period may be sufficiently long to permit a resurgence of disease. For weakly pathogenic species, persistence may be aided by the behavior of the extensive rhizomorph systems some of them form.

In unmanaged forests, longevity probably confers a survival advantage on all species, but it may be particularly important for the less pathogenic ones since the opportunity for them to acquire additional substrates may be more limited than for more pathogenic species. The latter may benefit, particularly in forests of susceptible species, by survival in disease gaps until a new crop becomes established. In forests which are managed intensively and are subject to selection cutting or regular thinning, longevity may no longer be a survival trait, at least for weakly pathogenic species, since a regular supply of stumps would be available for colonization.

Factors Affecting Growth of Rhizomorphs from Inoculum

The abundance, type, and distribution of rhizomorphs on a site are primarily determined by the *Armillaria* species present, but environment exerts a major influence through the effects of soil.

Variation Among Species

Whereas all *Armillaria* species form rhizomorphs to some degree in axenic culture, not all have been observed to do so in the field. No rhizomorphs have been reported for *A. tabescens* (Rhoads 1956, Rishbeth 1982, Ross 1970) although Rishbeth observed them on inocula buried in soil. In *A. luteobubalina*, they are either absent (Kile 1981, Shearer and Tippett 1988) or sparse under natural conditions (Pearce and others 1986, Podger and others 1978); other Australasian species, for example *A. limonea* and *A. novae-zelandiae* (Hood and Sandberg 1987), form rhizomorphs readily. *Armillaria hinnulea* forms rhizomorphs more prolifically than *A. luteobubalina*, but they are confined to root surfaces (Kile 1980b, Kile and Watling 1983). Among European and North American species, rhizomorph production is greater in *A. gallica* and *A. cepistipes* than in *A. ostoyae* and *A. mellea* (Gregory 1985, Guillaumin and others 1989a, Redfern 1975, Rishbeth 1985a). Information is lacking for some of the more recently described species such as *A. pallidula* and *A. fellea* (Kile and Watling 1988), but *A. sinapina* is reported to produce rhizomorphs abundantly in the field (Bérubé and Dessureault 1988). At the present time, information is insufficient to establish that the ability to produce rhizomorphs represents a continuum among species, but that may well be the case.

Morrison (1989) studied rhizomorph production by an array of species from Europe, Australasia, and North America using woody inocula buried in pots containing a mixture of forest soil, peat, and sand. While producing valuable information, such studies are not necessarily a reliable guide to field behavior. Thus, *A. luteobubalina* produced rhizomorphs more abundantly (fig. 4.1) under these circumstances than might have been anticipated from the field observations reported above. Podger and others (1978) reported similar results from pot culture, suggesting rhizomorph formation may be inhibited in the field by environmental conditions. For other species, observations under artificial conditions do coincide with field behavior (Gregory 1985; Redfern 1975; Rishbeth 1985a,b).

The growth habit of rhizomorphs in soil also varies among species; branching (fig. 4.1) is either monopodial or dichotomous (Morrison 1982b, 1989). This character may have ecological significance since Morrison (1989) found that species with dichotomously branched rhizomorphs tended to be more pathogenic than those producing monopodially branched rhizomorphs, but the distinction was not entirely consistent.

The Effect of Soil on Rhizomorph Growth

Most observations about soil have concerned its influence on the incidence and severity of disease, whereas the primary interest here is effect of soil on the fungus itself. The wide variety of soils associated with disease (Ono 1965, 1970, Rhoads 1956, Ritchie 1932, Shields and Hobbs 1979) suggests *Armillaria* species tolerate a fairly broad range of conditions.

Field observations on effects of soil on disease frequently conflict. Unfortunately, many are of limited value, and may be misleading, because they refer to *A.*

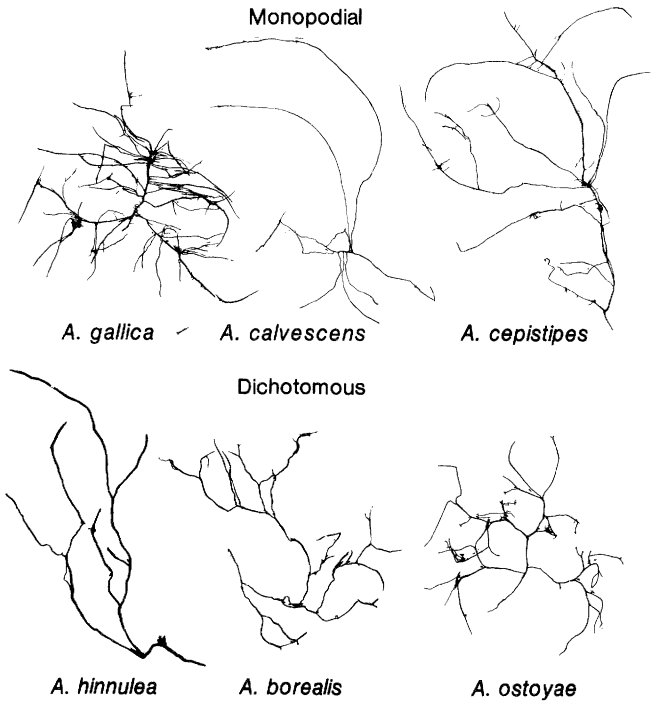


FIGURE 4.1—Variation in rhizomorph growth habit among *Armillaria* species. (Adapted from Morrison 1989).

mellea (sensu lato) when more than one species may be present. In these circumstances, differences in disease incidence due to the differing pathogenicity of the species involved may have been incorrectly attributed to soil factors. Similar misinterpretations may also arise through failure to appreciate the effects on disease development of the discontinuous distribution of inoculum.

Experiments in which woody inocula containing *Armillaria* isolates have been allowed to form rhizomorphs in soil (Gramss 1983; Morrison 1976; Redfern 1970, 1973, 1975; Rishbeth 1985b) confirm field observations that *Armillaria* can grow in a wide variety of forest and agricultural soils. Soil seems to exert a major influence on rhizomorph growth only under unusual or extreme circumstances. Thus, pure sand can partially inhibit rhizomorph production (Garrett 1956b, Redfern 1973, Rykowski 1984), whereas peat stimulates growth and branching (Redfern 1973). Certain tropical soils inhibit rhizomorph development (Dade 1927, Fox 1964, Rishbeth 1980, Swift 1968), and the paucity of rhizomorphs associated with damage by *A. luteobubalina* may also be soil-induced (Pearce and Malajczuk 1990a, Podger and others 1978).

A number of methods have been used to assess rhizomorph growth from woody inocula in soil. These include measuring the total length or dry weight of rhizomorphs and repeatedly measuring individual rhizomorphs. Rishbeth (1968) used the last method for testing the effect of temperature, and discussed some problems associated with this type of work.

Moisture

Working with *A. mellea (sensu lato)*, Garrett (1956b) and Redfern (1970) found soil moisture had no effect on growth within the ranges 40%-80% and 25%-75% of moisture-holding capacity, respectively. Growth of *A. luteobubalina* also occurs over a wide range of matric potentials (-0.0008 MPa to -7 MPa), but it is restricted below -0.6 MPa (which is roughly equivalent to 25% moisture-holding capacity). Seasonal drying may partly explain the paucity of rhizomorphs of this species in Australian soils (Pearce and Malajczuk 1990a). In Britain, Morrison (1976) concluded that seasonal drying may affect growth of *A. mellea (sensu lato)* in the upper soil layers.

Waterlogging may restrict growth at depth indirectly through the soil atmosphere (Rishbeth 1978a) and can prevent rhizomorph formation by inocula in pot experiments (Guillaumin and Leprince 1979). Despite the reservations already expressed about field observations, it is notable that *Armillaria* has rarely been reported from permanently wet soils with an appreciable peat accumulation. There is a single observation of *A. ostoyae* from Scotland (senior author and S.C. Gregory pers. comm.), and Hintikka (1974) commented that it seems to be largely absent from forested *Sphagnum* swamps, except where the peat is thin and the ground water is moving.

Temperature

The in vitro studies reported in chapter 3 provide a guide to the behavior of the fungus in soil, but caution should be exercised in extrapolating results since important differences may exist. For example, growth occurs at higher temperatures in agar culture than in soil (Rishbeth 1968).

Using woody inocula colonized by a suspension of basidiospores and by measuring growth directly, Rishbeth (1968) found the optimum temperature for rhizomorph growth through soil was about 22°C. Some growth occurred at 5°C and 28°C but none at 30°C. He concluded that rates of spread of about 1.5 m per year observed at sites in southern Britain, where the soil temperature at a depth of 15 cm averages 10°C, roughly corresponded with those determined from his experiments. Later, working with a number of isolates and species, Rishbeth (1978a) found the dry weight of rhizomorphs produced by inocula in soil was usually maximal at 20°C and minimal above 26°C and below

10°C. He suggested the lack of rhizomorph growth in forest soils at low elevations in tropical Africa (Dade 1927, Fox 1964, Swift 1968) may be due to high soil temperatures. By contrast, low temperatures may be limiting in many forest soils, particularly in the north temperate zone during winter (Rishbeth 1978a). However, the production of rhizomorphs from inocula involves two processes: initiation and growth. Initiation may occur over a more restricted temperature range than growth (Rishbeth 1968). Thus, pre-existing rhizomorphs may grow at lower temperatures than indicated by experiments in which rhizomorph production rather than growth is measured. Temperatures below 10°C may therefore be less restrictive than has hitherto been suggested. Although rhizomorph initiation may be curtailed in winter, growth of those initiated at higher summer temperatures may continue.

The effect of low temperatures receives some support from in vitro studies (Hintikka 1974, Pearce and Malajczuk 1990a, Rishbeth 1968), but as indicated earlier, they may not provide an entirely satisfactory guide to behavior in soil and further work is required.

Rishbeth (1978a) found variation in the effect of temperature on rhizomorph growth in soil among a worldwide selection of isolates, but there is little information for different species. Pearce and Malajczuk (1990a) tested growth of *A. luteobubalina* over a limited range of temperatures and found maximum growth at the highest temperature tested (20°C) with virtually no growth at 10°C. On agar, the optimum temperature for growth by this species was in the range 20-26°C, suggesting that it might be somewhat higher in soil. Also on agar, the more northern or high-altitude European species *A. borealis, A. cepistipes,* and *A. ostoyae* have a lower optimum for growth than the southern or low-altitude species *A. gallica* and *A. mellea* (Guillaumin and others 1989a). Thus, although there is some evidence for interspecific variation in the temperature relations of *Armillaria*, further work is required in soil.

Temperature may affect both the number and branching of rhizomorphs initiated from woody inocula. Redfern (1973) found that an isolate of *A. mellea (sensu lato)* initiated more rhizomorphs in soil at 25°C than at 15°C, and each system had a greater branching frequency at the higher temperature. This effect requires confirmation and further study with a range of species. The possibility that growth patterns may vary in response to seasonal variations in soil temperature is of particular interest and may have implications for infection and spread.

The studies on *A. luteobubalina* by Pearce and Malajczuk (1990a) demonstrated that rhizomorph behavior may be influenced by an interaction between temperature and moisture. This may well apply to other species, although the relative importance of the two factors may differ elsewhere.

pH

No body of field observations suggests that pH has a significant effect on *Armillaria*. Gard (1928) associated disease in Persian walnut with a reduction in lime content of the soil, and Rishbeth (1982) recorded killing by *A. ostoyae* on acidic soils but not at comparable sites where soil was alkaline. By contrast, he found *A. mellea* often killed trees on alkaline soils. In an inoculation experiment, Redfern (1978) found that infection by one isolate of *Armillaria* was significantly greater in an acidic soil than in an alkaline soil of similar sandy texture. However, in all these cases any pH effect may have been expressed through the host rather than through the pathogen. Other authors (Kawada and others 1962, Rhoads 1956) refer to killing on acidic soils but this probably only reflects the pH of most forest soils.

Experimental studies of pH effects are hampered by the difficulty of adjusting soil pH. In England, a succession of workers partly avoided the problem by taking advantage of a natural pH variation induced in uniformly sandy soil by differences in the depth of underlying chalk. In an initial experiment, rhizomorph production by a single isolate was greater at pH 7.5 than at pH 4.9 (Redfern 1970). Subsequently, more comprehensive work (Morrison 1974) with a range of isolates gave a variable response, with some isolates being unaffected. Further work by Morrison (pers. comm.) has shown that these differences were related to species. *Armillaria mellea* and *A. ostoyae* grew more in acidic than in alkaline soil, whereas *A. gallica* was either unaffected by pH or favored by alkaline soil. Rishbeth (1985b) tested three species in the same soils but detected no differences.

Inhibitory Substances

After several experiments with sterilized soil extracts, Swift (1968) attributed the absence of rhizomorphs from forest soils in Rhodesia (Zimbabwe) to a water-soluble inhibitor. Olembo (1972) found unsterile leachates of East African soils reduced the colonization of wood by *Armillaria*, but no further work has been done on this topic.

Organic Matter and Soil Nutrient Status

Accumulating evidence suggests soil nutrition affects rhizomorph growth. Rykowski (1984) confirmed the stimulating effect of peat (Redfern 1973) and observed a similar response to pine bark compost. Studying the

influence of various organic soil amendments on rhizomorph development, including peat, Guillaumin and Leprince (1979) obtained rather different results but nevertheless concluded that the surrounding medium affects growth. Morrison (1975) investigated the peat effect and demonstrated that rhizomorph growing tips absorb nutrients. He suggested that the nutrients available from a food base may be supplemented by uptake from soil, and that rhizomorph development may be related to soil nutrient status. Nutrient balance may also be important. Rykowski (1984) found growth in one soil was increased by application of potassium and reduced by nitrogen and phosphorus.

The Distribution of Rhizomorphs in Soil

Soil moisture affects the vertical distribution of rhizomorphs in soil. Morrison (1976) found rhizomorphs grow towards the soil surface, and has suggested this behavior is a response to the oxygen gradient in soil. Vertical distribution is probably controlled by seasonal desiccation of the upper soil layers and by oxygen and carbon dioxide concentrations lower down (Morrison 1976, Rishbeth 1978a). Hartig (1873b) noted rhizomorphs lie at about 10 cm depth, and Lawrence (1910) observed them in "great abundance from 3 to 18 inches below the soil surface." Later authors reported a concentration in the upper soil layers, generally within 10-20 cm of the surface (Day 1927b, Ono 1970, Redfern 1973). Where a humus layer is present, rhizomorphs are more common there than in the mineral soil below (Hintikka 1974, Singh 1981b), an interesting observation in view of the stimulating effect of peat on rhizomorph growth. The concentration of rhizomorphs in the upper soil layers may be important epidemiologically because of the greater vulnerability of trees to infections initiated on the root collar and proximal part of the root system compared to the deeper, more peripheral roots (Bliss 1946, Hintikka 1974, Patton and Riker 1959, Shaw 1980). Inoculum potential may also be greater than with a less stratified distribution.

These field data on rhizomorph distribution are most likely to have been contributed by species which form rhizomorphs capable of extensive growth through soil. Little information is available for species with rhizomorphs which are more closely associated with roots. Pearce and others (1986) found rhizomorphs of *A. luteobubalina* were present on infested sites at depths between 5 and 15 cm. Experimentally, *A. luteobubalina* produced rhizomorphs from woody inocula buried at 28 cm (Pearce and Malajczuk 1990a) although the number and total length were small. For species which spread mainly by root contact, it seems likely that the opportunity for infection and spread would be maximized by an ability to form rhizomorphs throughout the rooting depth of the host.

The horizontal distribution of rhizomorphs can be extensive. *Armillaria gallica* forms a network of rhizomorphs over the surface of living roots (Rishbeth 1985a). Redfern (1973), who probably observed the same species, suggested that rhizomorphs branch and anastomose to form extensive, complex networks which envelop both living trees and the food bases from which they originated. In one new plantation, Redfern (1973) estimated that rhizomorphs had spread up to 35 m in 37 years from adjacent, long-standing woodland infested by *Armillaria*. *Armillaria cepistipes* and other prolific rhizomorph-forming species may behave in the same way. In North America, Lawrence (1910) observed that rhizomorphs growing from infected raspberry canes formed a "network by frequently branching and rebranching", and Childs and Zeller (1929) referred to "a complete network of rhizomorphs about the larger roots" of orchard trees on fir-cleared land infested by a non-pathogenic species. Several authors have estimated the abundance of rhizomorphs in soil (Hintikka 1974; Hood and Sandberg 1989; Ono 1965, 1970; Rykowski 1984). Hintikka recorded 121 cm of rhizomorphs per 100 cm^2 of soil surface.

Inoculum Potential and Infection

Rhizomorphs represent extensions of inoculum, and are important in the infection, spread, and persistence of many *Armillaria* species. In a minority of species, rhizomorphs are absent or are only sparsely formed, and in these species infection is confined to points of contact between host roots and the inoculum. The infection process may involve epiphytic rhizomorphs or the transfer of mycelium, but the most important feature for the epidemiology of these species is the need for contact between host and inoculum. Infection also occurs in this way under certain environmental conditions which prevent or restrict rhizomorph formation. Garrett (1970) concluded from his experiments with rhizomorph-forming *Armillaria* species that inoculum size, distance between the inoculum and the host, and the influence of environment on the fungus were the major determinants of inoculum potential. However, where infection occurs at root contacts, only the first and last factors seem likely to be important.

This section primarily addresses those factors which affect disease development through their effect on inoculum potential. Chapter 5 describes the infection process in detail; here, attention is confined to the way in which infection occurs and its effect on the epidemiology of disease. The role of wounds in the successful establishment of infection is also considered.

Inoculum Potential

Little work has been done on inoculum potential since Garrett's classical experiments (Garrett 1956b, 1958) and none with species lacking rhizomorphs. Garrett experimented with model systems consisting of small woody inocula and potato tubers in soil. He found rhizomorph growth rate was related to inoculum size, and that the extent of infection in potato tubers increased with inoculum size and decreased with increasing distance between inoculum and tuber. Rhizomorph growth rate also declined with time, and he attributed this partly to nutrient depletion in the inoculum and partly to competition for nutrients between the main apex of a rhizomorph system and its subordinate branch apices. Rykowski (1984) recently confirmed Garrett's results, using larger inocula and Scots pine seedlings. He used indices derived from the number, length, and weight of rhizomorph systems produced from inocula, and the number of apices on those systems, to calculate the "potential infection threat" presented by inocula in various soils.

Although the concept of inoculum potential is simple and of considerable biological importance, it is difficult to envisage its application to individual trees since field situations are frequently complex. Inocula are rarely discrete, and infection often is not readily associated with specific point sources. Also, inocula vary in size from parts of individual roots to entire stumps.

The rhizomorph networks formed by some species may present an additional complication. The behavior of these systems requires study. Redfern (1973) suggested they may be relatively long-lived, being supported by a succession of food bases as they become available to different parts of the network, and the direction of nutrient flows changing to maintain the entire system from different sources. This is apparently inconsistent with experiments on translocation (Anderson and Ullrich 1982b, Schütte 1956) which have shown that it only occurs towards growing tips. Morrison (1975) found that nutrients absorbed by growing tips were not translocated towards the food base. These experiments do not represent the behavior of an entire network, however, and they are not inconsistent with the possibility that the direction of translocation within a rhizomorph in a network may vary with time. Anderson and Ullrich (1982b) commented that "if the (rhizomorph) base were converted to a sink for nutrients, as may be the case during fruiting or exhaustion of food reserves, rhizomorphs may transport nutrients from tip to base." This is supported by observations on severed rhizomorphs. Rhizomorphs forming part of a network and which are severed in situ initiate new rhizomorphs simultaneously from the cut ends, as do excised sections of large diameter rhizomorphs (Hintikka 1974, Redfern 1973, Rykowski 1984).

The principle of a fungal *corpus* consisting of a network of colonized stumps and rhizomorphs may apply equally well to species such as *A. mellea*, *A. ostoyae*, and *A. hinnulea* with more restricted rhizomorph-forming abilities. Roots may simply predominate over rhizomorphs in the network. However, some evidence indicates that, in contrast to *A. gallica* and *A. cepistipes*, rhizomorphs of *A. mellea* are short-lived and are produced in successive waves (Guillaumin and others 1989a) which suggest these species are unlikely to form persistent networks.

Clearly, much of the foregoing is speculative, but it is worth consideration since rhizomorph systems which behave in this way might create inocula consisting effectively of several stumps.

Despite this complexity, and notwithstanding the minute inocula used by Garrett (1956b) and Rykowski (1984) compared to substrates available naturally, there seems no reason to doubt the general applicability of the principle of inoculum potential to such large inocula. Inoculum potential is maximized where healthy roots and inoculum are in contact; where gaps are bridged by rhizomorphs, it diminishes with increasing distance between them. However, few detailed analyses of disease patterns in relation to the distribution of inoculum have been done. Understanding such patterns requires considerable knowledge of pathogen behavior in the circumstances of each outbreak, particularly the relative importance of rhizomorphs and root contacts as the means of spread in the species involved.

Shaw (1980) and Shaw and others (1976a) described a relatively straightforward situation in young ponderosa pine involving a single species, *A. ostoyae* (Shaw 1984), spreading essentially by root contact from discrete sources of inoculum. On the other hand, disease development following replacement of indigenous forest comprising many host species and more than one *Armillaria* species by a susceptible monoculture (MacKenzie and Shaw 1977) is much more complex. Under these circumstances, the pattern of mortality assessed on a single occasion (van der Pas 1981a) may be difficult to interpret (Roth and others 1979). MacKenzie and Shaw (1977) recorded decreasing mortality with increasing distance from infected stumps. Though such a pattern suggests the stumps were acting as the initial infection sources, interpretation of subsequent events in terms of inoculum potential is not possible. As suggested by Roth and others (1979), the effect could be caused by rapid, early killing within the rooting zone

of the stumps acting as inocula, followed by a slower rate of mortality outside this zone as growing roots or rhizomorphs bridge the gaps between potential hosts and inocula.

In another example, similar patterns of mortality among saplings around eucalypt stumps infected by *A. luteobubalina* (Pearce and others 1986) may simply have reflected the time when the developing sapling root systems made contact with stump roots. Alternatively, disease centers in young conifers (probably caused by *A. ostoyae*) show reduced extension rates because of increasing host resistance (Redfern 1978) rather than declining inoculum potential with increasing distance from a point source.

On a large scale, the effect of an increase in inoculum can be appreciated readily. Forestry operations such as clear-felling, selective cutting, thinning, the treatment of indigenous crops with herbicides, or events such as fire provide opportunities for a massive increase in inoculum (Kile 1980b, Pronos and Patton 1978, Rishbeth 1972b, Shaw and others 1976a, Swift 1972, van der Pas 1981b). For pathogenic species, more inoculum typically results in more disease. Thus, *A. luteobubalina* causes disease in unlogged eucalypt forest (Kile 1983b), but the greatest incidence and severity of disease is associated with logging (Kellas and others 1987, Pearce and others 1986). Concomitantly, natural regeneration or planting repositions hosts relative to the inoculum. Thus, in former disease centers which are devoid of hosts, or in plantation systems where trees are planted beside stumps which may subsequently become colonized, the distance between potential hosts and inoculum may be small.

Physical disturbance of the soil by logging, plowing, scarifying, or even planting may sever rhizomorph networks, which respond by initiating new growing tips from the cut ends. Besides increasing the amount of inoculum and perhaps the availability of new and more susceptible hosts, harvesting disturbance can also stimulate the production of rhizomorph growing tips and locally increase the chance of infection (Redfern 1973, Rykowski 1984, see chapter 11).

Inorganic fertilizers may influence inoculum potential through the soil environment. The effect of some macro-nutrients on rhizomorph production by inocula in soil has already been mentioned (Rykowski 1984). The inoculum may also be directly affected. Work by Azevedo (1970-71), Garrett (1953, 1970), and Rykowski (1976a) suggests the possibility that crop fertilization might increase inoculum potential by changing substrate quality when roots with an enhanced nutrient status eventually become inoculum. Both of these interesting possibilities merit further study.

To assess the need for control in *Armillaria*-infested areas (see chapter 11), forest managers must estimate the inoculum potential of the species present in addition to knowing their pathogenicity and distribution. However, even in the simplest situation involving only one species, there seems little possibility that the inoculum potential of *Armillaria* on a site could be assessed by casual observation. For example, although it has been used for modeling purposes (see chapter 10), stump size may be a poor guide unless colonization is complete. The circumstances under which complete colonization may be achieved include the invasion of living, susceptible conifers by highly pathogenic species, the colonization of freshly felled conifers by growth of the same species from root lesions, and the colonization of healthy conifer stumps by species capable of forming extensive rhizomorph systems. In hardwoods, however, colonization may be restricted in those species which tend to regrow after cutting (Rishbeth 1972b). In some eucalypts (Kile 1980b), and possibly oaks, the heartwood is resistant to decay and remains uncolonized. Pearce and others (1986) found a significant relationship for *A. luteobubalina* between an estimate of how much inoculum was provided by individual, infected stumps and mortality in nearby saplings; assessments like this are unlikely to be feasible in commercial forestry, however.

A reduction in inoculum potential or the prevention of inoculum buildup provides the basis for many control measures (see chapter 11). Under natural conditions, the amount of *Armillaria* inoculum on a site may be reduced by competition from other fungi and by fire. In the case of wood-rotting fungi which are also parasites, such as *Phellinus weirii* (Morrison and others 1988) and *Heterobasidion annosum* (Greig 1962), competition is not beneficial; but some saprophytic decay fungi are also able to compete successfully and may be useful for biological control (Pearce and Malajczuk 1990b, Rishbeth 1976). The soil-inhabiting fungus *Trichoderma viride* may exert a degree of control which can be enhanced by soil fumigation (Bliss 1951; Garrett 1957, 1958; Ohr and others 1973). Fire may kill rhizomorphs in soil (Hood and Sandberg 1989), but its effects on inoculum survival and subsequent rhizomorph activity are unknown.

A massive inoculum is not a prerequisite for infection if the distance between inoculum and host is minimal. Many experiments have demonstrated that successful infections can be established on small trees by means of small woody inocula, some weighing as little as a few grams (Patton and Riker 1959, Rykowski 1984). This has particular relevance for control by inoculum removal since root fragments inevitably remain after destumping and root raking operations (Morrison and others 1988). Although a high level of control can be

achieved by destumping, certainly a level which would return an infested site to normal productivity, residual root fragments may nevertheless permit the re-establishment of disease. Damage may be confined to a few early losses, but it could be extended by secondary, tree-to-tree spread (Rykowski 1984).

Although small and large inocula may both cause infection, Rykowski (1984) has suggested that each represents a different type of threat. In experiments with small, woody inocula, rhizomorph production per unit volume of inoculum was inversely related to total inoculum volume. This suggests that rhizomorph production is delayed until the substrate has been fully colonized and certain nutritional requirements have been met (Benjamin and Newhook 1984b, Garrett 1953, Patton and Riker 1959, Rykowski 1984). Rykowski concluded that whereas in small substrates the phases of colonization, rhizomorph production, and exhaustion are accomplished rapidly, the same process takes longer in larger inocula. He argued that stumps may behave in the same way, presenting short-term and long-term infection threats, respectively.

Infection

As already discussed, rhizomorphs are formed in soil to a greater or lesser extent by most *Armillaria* species; the absence of rhizomorphs is apparently uncommon among species in the genus. In some species, they are restricted to root surfaces or to the close proximity of roots, whereas others form abundant rhizomorphs which ramify freely through soil. Without rhizomorphs, infection is confined to points of contact between host roots and the inoculum; with increasing rhizomorph production, infection can also take place at greater distances from the inoculum.

Because rhizomorphs are often abundant, much of the early literature from temperate countries emphasized the importance of rhizomorphs growing freely through soil as a means of spread. Indeed, some authors considered them essential (van Vloten 1936). However, a number of authors either observed infection at root contacts (Kawada and others 1962, Přihoda 1957, Zeller 1926) or inferred its occurrence from their observations (Marsh 1952, Molin and Rennerfelt 1959). Working in black currant plantations, Marsh (1952) found the pattern of disease spread was best explained by root contact infection rather than by rhizomorphs growing in soil unoccupied by roots. Molin and Rennerfelt (1959) concluded that spread occurs mainly by root contact, and rhizomorphs only play a secondary role except over distances less than 1 m. In Czechoslovakia, Přihoda (1957) referred specifically to infection of Norway spruce both by rhizomorphs and by the transfer of mycelium at root contacts where rhizomorphs were absent. He commented that although soil rhizomorphs were present on one site, they were sparse and weak and the bulk of infection was by mycelium transfer. He discussed the possibility that rhizomorph formation might be inhibited by alkaline soils, but he concluded that soil was unimportant and that some "forms" of *Armillaria* do not produce rhizomorphs whereas others do so abundantly.

Without our present understanding of *Armillaria* speciation and ecology, earlier authors did not appreciate the difference between spread of the more pathogenic species among susceptible hosts and the spread of less pathogenic species on stumps and weakened trees. Přihoda's comments (1957) were therefore particularly percipient. These European observations of spread by root contact probably referred to either *A. ostoyae* or *A. mellea*, which are pathogenic and form fewer rhizomorphs than the weakly pathogenic species *A. gallica* and *A. cepistipes* (Guillaumin and others 1985, 1989a; Rishbeth 1985a). Abundant rhizomorph production by the latter species may also prompt misinterpretation where they occur with pathogenic species if it is assumed that any rhizomorphs observed in soil are those of the disease-causing species.

For species such as *A. tabescens, A. hinnulea,* and *A. luteobubalina* in which rhizomorphs are either absent or confined to root surfaces, infected roots must be in contact with potential hosts, or very close to them, for infection to occur (Kile 1980b, 1981; Kile and Watling 1983; Pearce and others 1986; Shearer and Tippett 1988). Nevertheless, interlocking root systems can provide highly effective pathways for spread by pathogenic species among susceptible hosts. Surveying dieback in messmate stringybark and mountain ash associated with *A. hinnulea* (Kile 1980b, Kile and Watling 1983), Kile (1980b) found that 74% of living trees had infections or epiphytic rhizomorphs on the root system. By contrast, species which form extensive rhizomorph systems, such as *A. gallica* and *A. cepistipes,* are not restricted in this way. *Armillaria mellea* and *A. ostoyae* have a lesser ability to form rhizomorphs in soil than *A. gallica*, but they are not confined to root surfaces and these species may occupy an intermediate position. In ponderosa pines, *A. ostoyae* spread between roots near to each other as well as at contacts (Shaw 1980).

In New Zealand, free-growing rhizomorphs are common in soil where both *A. limonea* and *A. novae-zelandiae* are present (Hood and Sandberg 1987), but the relative contribution of each species to the rhizomorph population is unknown. However, both species readily produce rhizomorphs in pot culture (Benjamin and Newhook 1984b), so it is likely that rhizomorph spread is important in both cases.

Recent work has suggested a relationship between rhizomorph production and pathogenicity among some European species. The more pathogenic species tend to produce smaller rhizomorph systems than less pathogenic species (Gregory 1985; Guillaumin and others 1985, 1989a; Redfern 1975; Rishbeth 1985a). Some evidence indicates the relationship may also apply to North American and Australasian species (Morrison 1989). Further information, particularly about rhizomorph production, is required for many of the more recently described species, but differences appear to be large (Gregory 1985, Redfern 1975) and may have considerable ecological significance. For a weak pathogen, a strategy involving a wide dispersion of inoculum offers the advantage of position when potential food bases become available. Thus, weakly pathogenic species which form extensive rhizomorph systems and infest roots in a network of rhizomorphs are able to exploit this advantage in the acquisition of substrates, which may consist of stumps or living trees with declining resistance. More pathogenic species, by contrast, do not require such a strategy and are able to spread among susceptible hosts through root contacts.

It follows from this discussion that spread in pathogenic species is likely to be influenced more by factors affecting the distribution of tree roots than by those which affect rhizomorph development. Thus, for purposes of disease management, pathogenic species in North America and Europe should perhaps be considered to have a greater affinity with *Phellinus weirii* or even *Heterobasidion annosum* than they traditionally have been.

Before our present understanding of speciation and pathogenicity in the genus, considerable debate focused on the environmental conditions required for infection and on the need for infection courts provided by root wounds or debilitated roots. The distinction is important since otherwise healthy roots which are physically wounded, perhaps by abrasion against stones, by animals, or by harvesting machinery, differ greatly from roots debilitated by, for example, poor soil aeration.

From the many inoculations which have been done on wounded roots, little doubt remains that infection can take place through wounds; but their importance as natural infection courts, however, has not been established clearly. Dimitri (1969) concluded that although infection in Norway spruce can take place through healthy, undamaged roots, it occurs primarily through wounds and dead roots. Buckland (1953) reported that he was unable to detect infection through healthy bark in vigorous Douglas-fir, observing it only in roots which had been mechanically damaged or physiologically weakened. Hintikka (1974) believed root collar injuries caused by snow bend promoted rhizomorph penetration at this point. However, it is difficult to determine by observation alone the role of wounded or stressed roots in the establishment of infection. In one of the few inoculation experiments designed to test the effect of wounding, Weaver (1974) found that it increased the number of isolates of *A. tabescens* which were able to infect peach roots. Invasion was also more extensive in injured roots. More recently, Whitney and others (1989b) found wounding increased infection in balsam fir inoculated with *A. ostoyae*.

Evidence from natural disease outbreaks, and the ease with which unwounded trees can be infected in inoculation experiments, suggest that, at least for the more pathogenic species, wounds are unlikely to increase the success of infection. Wounds and debilitated roots could be important infection courts for less pathogenic species such as *A. gallica*, but no evidence supports this. Gregory (1985) showed that the length over which rhizomorphs became attached to the host surface was greater for species of low pathogenicity than for those of high pathogenicity. This could be expected to provide weakly pathogenic species with a greater opportunity to encounter wounds than would be available to pathogenic species.

Conclusions

Wood, mainly tree roots, provides the major source of inoculum for *Armillaria*. Many older observations of disease supported the view that hardwoods provide a superior substrate for *Armillaria* than conifers. In general, little experimental evidence substantiates an intrinsic difference between the two substrates but stumps of broadleaved trees may exhibit greater longevity as inoculum. Some *Armillaria* species may subsist better on particular food base species, but there is no evidence for substrate specialization. However, a degree of ecological specialization is known for some north-temperate species.

All species form rhizomorphs in culture, and almost all do so in forest soils, but they vary greatly in the amount of rhizomorph growth. Some species are epiphytic or restricted to the close proximity of roots, whereas others grow freely through soil, forming networks which link both colonized stumps and living trees. Infection is probably caused by rhizomorphs in most species, either at contacts between host roots and the inoculum or at some distance from the inoculum. For species lacking rhizomorphs, or where soil conditions prevent their formation, infection is restricted to contacts and occurs by the transfer of mycelium. Species with epiphytic rhizomorphs are similarly restricted, but infection can be either by mycelium transfer or by rhizomorphs. The relative importance of

the two methods for these species is unknown. No information is available about the influence of soil on infection by mycelium transfer.

The environment can have a major effect on *Armillaria* through the effects of competing fungi on survival in woody substrates and through the influence of soil on rhizomorph growth. The fungus can grow in soils derived from a wide variety of lithologies, but more fertile soils may be particularly favorable since nutrient uptake from the soil may supplement nutrients from the food base. Soil moisture, temperature, and pH all affect rhizomorph growth, and there is some evidence for an interaction between moisture and temperature which may also be important. Species differ in their response to temperature and pH, but little information is available.

The inoculum potential of *Armillaria* is influenced by the amount of inoculum, by the distance between the inoculum and the host, and by environmental effects. Forestry operations such as felling and thinning increase inoculum on a site, but patterns of mortality should not be interpreted simply in terms of inoculum potential. Interaction between, among other things, the amount and distribution of inoculum, the method of spread by the *Armillaria* species involved, and root system development by the host may be equally important.

The more pathogenic *Armillaria* species may produce smaller rhizomorph systems than less pathogenic species. Further information is required, particularly for more recently described species, but such a tendency may have considerable ecological significance. Thus, the extensive rhizomorph systems produced by weakly pathogenic species may represent a strategy for the wide dispersal of inoculum in order to gain the advantage of position when potential substrates become available. By contrast, the interlocking root systems of susceptible hosts may provide an effective means of spread for more pathogenic species, and obviate the need for extensive rhizomorph systems.

Wounds may be important infection courts for weakly pathogenic species, but they are unlikely to increase the success of infection by more pathogenic species.

CHAPTER 5

Infection, Disease Development, Diagnosis, and Detection

Duncan J. Morrison, Ralph E. Williams, and Roy D. Whitney

The first comprehensive description of Armillaria root disease, including the causal fungus and its life cycle, was made by Robert Hartig (1874). He recognized that *Rhizomorpha fragilis* Roth with its two chief forms, *R. subterranea* and *R. subcorticalis*, composed part of the mycelial body of *Agaricus (Armillaria) melleus*. *Rhizomorpha subterranea* and *R. subcorticalis* were the binomials applied to the cylindrical brown to black mycelial strands found in soil and on root surfaces and the flattened white to cream colored mycelial felts (fans) found between the bark and wood of hosts, respectively. Hartig observed the basidiomes of *A. melleus* developing on rootstocks with *R. subcorticalis* under the bark and on rhizomorph apices in soil. He also described infection and disease development in several conifer species.

Since Hartig's work, more than 600 species of woody plants have been recorded as hosts of *Armillaria* species (Raabe 1962a). The infection process and disease development have been described for several hardwood and coniferous hosts. A wide variety of symptoms, signs, and host responses resulting from disease have been recorded, reflecting the wide host and geographical ranges and number of *Armillaria* species. This chapter describes the infection process and disease development in photosynthesizing (green) plants, the symptoms and signs on diseased plants, and how these symptoms can be used to detect Armillaria root disease in forests and orchards.

The Infection Process and Disease Development

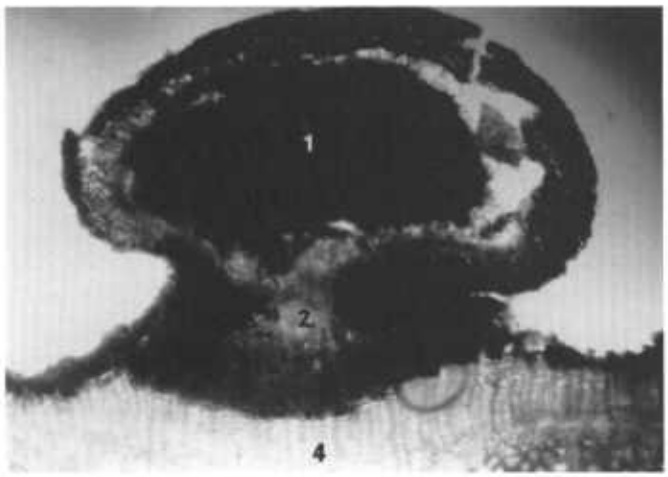
The Infection Process

Thomas (1934) defined infection by Armillaria root disease as penetration of the fungus into the host, with or without subsequent colonization. The roots of woody plants may be infected following contact between a suscept root and a rhizomorph or diseased root. Although many records document many different hosts being attacked (Raabe 1962a), the literature on the infection process is sparse. All detailed studies of the infection process predate acceptance by pathologists that *Armillaria mellea sensu lato* represents many species. Diverse host responses and sometimes conflicting reports about the same host are evident in accounts of disease occurrence. These apparent discrepancies may be partly attributable to different *Armillaria* species having been involved. Current knowledge of the geographical distribution and host preferences of *Armillaria* species helps clarify the identity of *Armillaria* species reported in early studies. For example, the *Armillaria* on pine (Hartig 1874) is probably *A. ostoyae* (H. Marxmüller pers. comm.) and Thomas' (1934) studies on hardwood trees probably involved *A. mellea sensu stricta*.

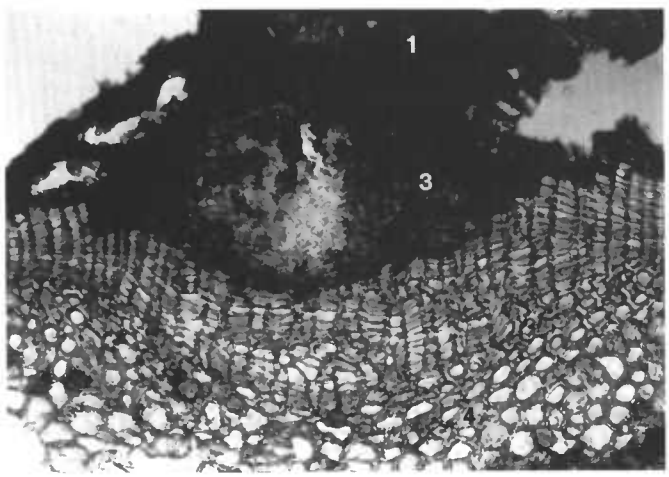
The first account of the *Armillaria* infection process was given by Hartig (1874). He wrote, "The killing of roots is brought about by *Rhizomorpha fragilis* which bores into the root, spreads out in all directions as *R. subcorticalis* and thus from the point of attack continually approaches the root stock until this is reached."

General agreement exists among the detailed studies of coniferous (Day 1927b, Rykowski 1975, Woeste 1956) and hardwood hosts (Guillaumin and Rykowski 1980, Thomas 1934) about the infection process by rhizomorphs. A rhizomorph becomes attached to a root initially by hardening of the mucilagenous substance which covers its growing tip. Then, single hyphae developing from the rhizomorph tip and penetrating the outer layer of cork cells anchor the rhizomorph to the root. On suscepts with smooth bark, branches which will form the root-penetrating rhizomorph develop at points of firm contact with the root surface. The branches originate in the inner cortical cells of the rhizomorph when hyphae divide and spread laterally. These hyphae force their way through the outer cortical cells of the rhizomorph and emerge as a branch. Branches may be numerous and always develop on the side of the rhizomorph contacting the host (Thomas 1934).

Thomas (1934) studied how *Armillaria* infected fleshy and woody roots of susceptible and resistant hosts. Penetration of the rhizomorph was essentially the same for both groups. The lateral branch, acting as a unit, not as individual hyphae, begins to penetrate by mechanical force. The host cork cells under the rhizomorph are pushed in and slightly compressed (figs. 5.1A,B). At this stage, tissues below the cork cells appear disorganized, which is attributed to secretions from the rhizomorph. Penetration continues by a combination of chemical and mechanical means. Beneath the cork, the rhizomorph branches spread laterally and radially into bark tissues. The descriptions of this process by Day (1927b) and Woeste (1956) indicate that more chemical destruction of tissues occurs in conifers than in hardwoods. Enzymatic breakdown of suberin may also be involved in bark penetration (Swift 1965, Zimmermann and Seemüller 1984).



A



B

FIGURE 5.1 — Penetration of the bark of a walnut root by *Armillaria mellea*. A: Subterranean rhizomorph with a developing lateral branch; B: Infection wedge penetrating host bark (1- rhizomorph; 2- rhizomorph branch; 3- infection wedge; 4- host bark). (J.J. Guillaumin)

In suscepts with scaly bark, the rhizomorphs (*R. subterranea)* run tangentially under bark scales becoming *R. fragilis* (Woeste 1956); that is, white strands without a rind. Rhizomorphs may emerge successively from beneath bark scales along a root. *Rhizomorpha fragilis* (as *R. subcorticalis*) may penetrate the bark scales and develop infection wedges beneath each one. Cell walls turn brown and cell contents become disorganized some distance from the infection wedge.

Day (1927b), Thomas (1934), and Woeste (1956) concluded that rhizomorphs of *Armillaria* need neither wounds nor anatomical points of weakness to attack healthy, vigorously growing suscepts. However, root injuries caused by stones and wind-induced root movements, wounds made by insects and scarification equipment, and rootlets killed by excessive moisture could all serve as infection courts (Basham 1988, Dimitri 1969, Kile 1981, Rizzo and Harrington 1988b, Whitney 1961). Two years after inoculation with *A. tabescens*, most isolates had infected injured roots of peach, whereas only a few isolates had infected uninjured ones, and invasion of injured roots was usually more extensive (Weaver 1974).

Zeller (1926) described infection of suscept roots by mycelial transfer across points of contact with diseased apple roots. He suggested that infection of the suscept root begins when its healthy bark is acted upon by toxic substances produced by *Armillaria* in the contacting diseased root. Shallow brown spots appear in the bark's outer parenchyma, and these eventually coalesce. Flakes of dead cork are sloughed as new cork layers are formed. *Armillaria* mycelium was not found in the spots until two or more plates of cork had been sloughed. Eventually, the fungus reaches the cambium and a canker develops. Conifers may become infected in a similar manner (Morrison unpubl.). Initially, mycelial fans of *A. ostoyae* grow in a root's outer bark. As the area of colonized bark increases, mycelial fans penetrate to the cambium. Bark tissue becomes necrotic in advance of the mycelial fans.

Host Response to Infection

Host responses to Armillaria root disease fall into three categories: exudate production, meristematic activity, and biochemical interaction. At the biochemical level, fungal infection involves an interaction between compounds already present in the host or induced by infection and extracellular fungal metabolites. These biochemical interactions are discussed in chapter 3. Here, responses involving meristematic activity and exudates are discussed.

Meristematic activity leading to cork and callus formation and, frequently, adventitious roots is a common

host response to *Armillaria* infection on roots and at the root collar. Most descriptions of the infection process by rhizomorphs indicate that all living, vigorous suscepts responded to bark penetration by forming one or more secondary cork layers beneath the point of penetration. Thomas (1934) noted that in resistant hosts the lesion produced by initial penetration was walled off by the secondary periderm; this cork layer then widened with root growth. In susceptible hosts, penetrating rhizomorphs breach these secondary cork layers. Rykowski (1975) observed similar reactions in Scots pine roots. On some roots, the penetrating rhizomorph reached the cambium whereas on others secondary cork isolated the infecting mycelium from living host tissues and caused infected bark to be sloughed (fig. 5.2). Observations on plum rootstocks showed that their resistance to *A. mellea* was mainly due to post-infection reactions, because the success rate in penetration by the fungus was similar for susceptible and resistant rootstocks (Guillaumin and others 1989b). Mycelial fans in the bark and sapwood grew considerably less in resistant rootstocks, and the slower growth was associated with pink or purple discoloration of bark and wood tissues surrounding lesions (fig. 5.3).

FIGURE 5.2 — *Armillaria ostoyae* lesion on a Douglas-fir root in which secondary bark has isolated the infecting mycelium (1-xylem; 2-bark; 3-infected bark). (D.J. Morrison)

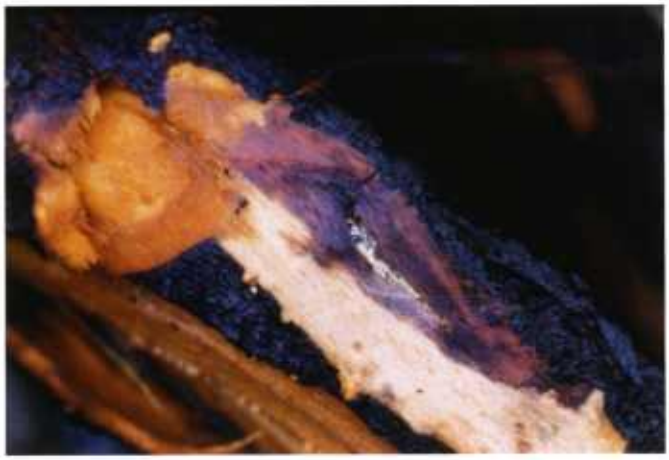


FIGURE 5.3 — *Armillaria mellea* lesion on a resistant *Prunus* sp. rootstock. Note purple discoloration of bark around the lesion. (J.J. Guillaumin)

Perhaps this post-infection reaction, which only occurs in living tissues, kills the mycelium thus preventing disease development.

Many hosts respond to *Armillaria* attack by exuding resin, gum, or kino. In hardwood hosts in which a pathogenic *Armillaria* species had penetrated to the cambium, Thomas (1934) observed that the xylem became brown ahead of penetrating hyphae. This reaction appeared to result from a gummy deposit in the vessels, perhaps secreted from the affected cells or a product from their walls. Resin production in pines was confined to areas of bark where mycelium had penetrated to the cambium and was not associated with ectotrophic spread in bark scales (Redfern 1978). On 5-year-old Corsican pine, the resin response was much more vigorous on trees inoculated with *A. mellea* than with *A. ostoyae* (Rishbeth 1982). Copious resin formed beneath bark tissue infected by *A. mellea*, forcing the tissue away from the root wood. Some mycelial sheets of *A. mellea* containing the resin were not viable, while those of *A. ostoyae* appeared to be unaffected (Rishbeth 1982).

The effects of conifer resin or resin components on *Armillaria* growth in culture vary. Pinene inhibited growth of *A. ostoyae* and *A. gallica* (Entry and Cromack 1989) and volatiles in Scots pine oleoresin reduced the growth of *Armillaria* by one-half (Rishbeth 1972a). However, powdered wound resin from ponderosa pine, when added to malt extract agar, significantly increased *Armillaria* growth compared to the basic medium (Shaw and Roth 1976).

Similarities may exist between *Heterobasidion annosum* (Fr.) Bref. and *Armillaria* in how oleoresin affects mycelial growth. Oleoresin did not affect *H. annosum* growth in agar culture. Prior (1976) suggested that resin-impregnated root wood of Corsican pine was a physical impediment to the fungus, reducing mycelial growth rate by more than one-half compared to non-resinous roots. Rykowski (1975) observed that resin-soaked wood and callus around root lesions on Scots pine limited spread of *Armillaria*; hyphae were not found in the resinous wood. Similarly, in roots of young, vigorously growing Douglas-fir trees, the host checked infections by laying down a callus and resin barrier (Buckland 1953), thus forming a latent canker (fig. 5.4). Cankers were 2-3 cm long, covered with resin, bounded by callus tissue, and often several years old. Within cankers, *A. ostoyae* either remained dormant or grew into the woody core of the root. Lesions at the root collar developed from one or more diseased lateral roots (Day 1927b). After killing the cambium at the root collar and on the lower bole, further spread of *Armillaria* was checked and callusing occurred (figs. 5.5A,B). In conifer roots, a central column of decay caused by *Armillaria*

FIGURE 5.4 — *Armillaria ostoyae* lesion on a Douglas-fir root. Note callusing at the margin of the lesion. (G.W. Wallis)

was compartmentalized by a barrier zone consisting of complete rings of resin ducts and parenchyma or numerous resin ducts separated by tracheids (Tippett and Shigo 1981).

In eucalypts, the development of decay in the roots and lower bole reflects differences in their response to *A. luteobubalina* (Shearer and Tippett 1988). Roots of jarrah often had bark lesions which were confined by new layers of periderm. Barrier zones formed in the xylem as a result of infection and were obvious boundaries between xylem produced before and after infection (fig. 5.6). *Armillaria luteobubalina* often girdles wandoo at the root collar because the tree does not resist tangential spread of the fungus in the inner bark. In contrast, callus tissue formed by jarrah and messmate stringybark restricted tangential spread, causing inverted V-shaped lesions (Kile 1981, Shearer and Tippett 1988). On citrus trees attacked by *A. tabescens*, living roots had bark lesions up to 30 cm long, some of which were delimited by callus (Rhoads 1948).

Adventitious roots arising from callus tissue (fig. 5.7) may compensate for roots killed by Armillaria root disease (Cooley 1943, Kile 1980b, Riggenbach 1966, Rishbeth 1985b).

The incidence of mortality by Armillaria root disease often decreases with increasing plant age, particularly in conifers (Buckland 1953, Gibson 1960, Johnson and others 1972, MacKenzie 1987). This decrease usually is attributed to increased host resistance with age, which could be associated with physiological or biochemical changes in the host. In lodgepole pine, resin production increases with age until about 50 years (Shrimpton 1973). The ability of conifers to form callus where lesions form on lateral roots and the root collar increases between age 5 and 20 years (Johnson and others 1972).

Post-Infection Development

Post-infection development of Armillaria root disease in a host root system depends upon the susceptibility, size, and age of the host (see below), the pathogenicity (see chapter 6) and inoculum potential (see chapter 4)

A

B

FIGURE 5.5 — A: *Armillaria ostoyae* lesion on the lower bole of a 21-year-old Douglas-fir. Note loosened bark and blackened resin. B: Cross section through lesion in (A). Note active callusing of lesion. (D.J. Morrison)

FIGURE 5.6 — *Armillaria luteobubalina* lesion on a greatcone banksia root. One side of the root was killed by the fungus. The area of xylem discoloration is bounded by a barrier zone. (Figure 5E from Shearer and Tippett 1988)

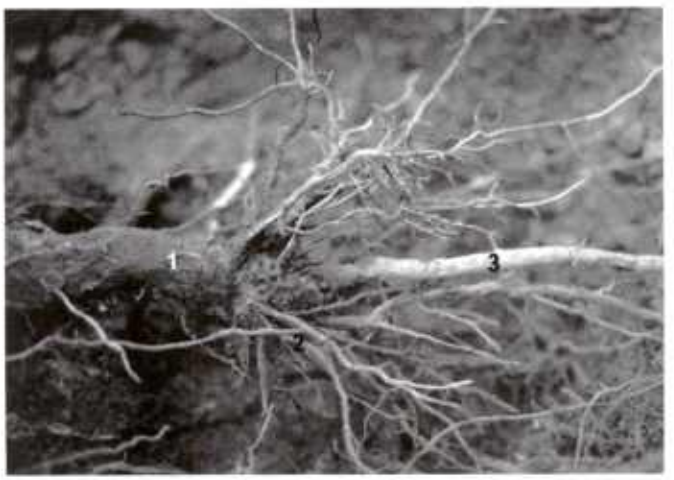


FIGURE 5.7 — Adventitious roots arising from a callused *A. ostoyae* lesion on a Douglas-fir root (1- living root; 2- adventitious roots; 3- *A. ostoyae*-killed root). (D.J. Morrison)

of the fungus, and the influence of environment on host-fungus interaction (see chapters 7, 8). In susceptible hosts, the rhizomorph which causes infection penetrates to the cambium, becomes *R. subcorticalis*, and spreads laterally in all directions through the cambial zone (Woeste 1956). Growth of mycelial fans in the outer bark may precede that in the cambium; that is, growth is ectotrophic. The extent of ectotrophic growth is variable. In messmate stringybark mycelium of *A. luteobubalina* in the outer bark was up to 1 m ahead of cambium infection (Marks and others 1976). In Scots pine (Redfern 1978), mycelium of *A. ostoyae* was only 2 cm ahead of established infection proximal to the infection point. As occurs with the penetrating rhizomorph branch, mycelial fans act as a unit, and host tissues are affected ahead of them. Schmid (1954) described the invasion of spruce bark by *R. subcorticalis*. In the xylem, mycelium penetrates the rays and spreads from them laterally into the xylem elements (Dade 1927, Woeste 1956). Continued killing of host tissues in the cambial zone girdles the root. The fungus spreads distally and proximally from the point of infection, and on reaching the root collar it spreads to other primary roots.

The location of infections is an important factor in disease development. Whether the result of contact with rhizomorphs or diseased roots, infections at the root collar or on the tap root (if present) usually kill the host more rapidly than infections on lateral roots (Barss 1913, Gadd 1930, Shaw 1980). However, infections at either location may be lethal (Rhoads 1948). On sapling and pole-sized ponderosa pines, Shaw (1980) found that rhizomorph-initiated infections on lateral roots were common, although the fungus rarely advanced proximally more than a short distance from a girdling root lesion. *Armillaria* infections on lateral roots may have failed to spread proximally because of host response, because rhizomorphs and distal portions of small roots may have provided inadequate inoculum potential, or both. Lethal attacks occurred high on the tap root or on the root collar. Similar observations were made on young Douglas-fir (Buckland 1953), on red pines and eastern white pines, and on white spruce (Patton and Riker 1959). Rykowski (1975) described the development of disease in the root systems of Scots pines, showing seven distinct patterns of infection.

Where rhizomorphs cannot establish progressive infections or for species which do not form them in forest soils, infections develop at contacts between healthy and diseased roots. Contacts are more likely to occur on lateral roots than at the root collar. On cacao (Dade 1927), citrus (Rhoads 1948), Douglas-fir (Morrison 1981), and eucalypts (Pearce and others 1986, Podger and others 1978, Shearer and Tippett 1988), infections originating this way on lateral roots spread to the root collar (fig. 5.8) and then to the tap root and other lateral roots, eventually girdling the trunk.

When *Armillaria* girdles a root, the portion distal to the infection is colonized rapidly by mycelial fans growing in the cambium (Redfern 1978, Shaw 1980). Redfern (1978) observed maximum spread of 110 cm (mean 62 cm) in 10 months in inoculated roots which had been severed.

Effects on the Host

In agricultural crops, Armillaria root disease may reduce the quantity and quality of produce prior to a plant's death. In forest crops, the disease may reduce height and diameter growth, cause decay of the bole, or cause death of the host, directly or indirectly.

FIGURE 5.8 — *Armillaria ostoyae* infection spreading along a Douglas-fir root (1- mycelial fan in outer bark; 2- bark necrosis in advance of the mycelial fan; 3- cambial necrosis). (D.J. Morrison)

Reduction in height and diameter increment is a consequence of partial killing of the host's root system. Ten-year-old radiata pine showed a highly significant difference in cumulative mean increment between healthy trees and those with more than 65% of root collar circumference showing symptoms of *A. limonea* or *A. novae-zelandiae* (Shaw and Toes 1977). Diameter growth of 70- to 80-year-old Norway spruce affected by Armillaria root disease was reduced from one to six times compared with healthy trees (Sokolov 1964). Annual growth increment of diseased 80- to 120-year-old Norway spruce was about one-half that of healthy trees (Molin and Rennerfelt 1959). Kile and others (1982) observed reduced growth in messmate stringybark with over 25% of their root collar circumferences infected by *A. luteobubalina*. Norway spruce (110 years old) which were classified as heavily infected by *Armillaria* had wider growth rings early in the rotation than trees which were healthy or lightly infected (Hřib and others 1983). This suggests that faster growing trees become infected earlier and more frequently due to their more extensive root system and greater probability of contacting inoculum (Bloomberg and Reynolds 1985).

Later in the rotation, ring widths of trees in the two highest infection classes were 1 mm or less compared to 3 mm in uninfected trees (Hrib and others 1983). MacKenzie (1987) estimated volume loss of 6%-13% due to lethal and sublethal infection over a 28-year rotation of radiata pine. Growth loss due to *A. ostoyae* in 80- to 100-year-old Douglas-fir was measured on trees stratified by the proportion of the root collar showing resinosis (Bloomberg and Morrison 1989). Growth during 5-year periods, expressed as a percentage of the stem volume at the start of each period, decreased significantly as resinosis increased due to colonization of the root system. In recently killed trees and in those with more than 50% basal circumference showing resinosis, growth began to decline 30 years previously. The volume increment of these trees during the last 5-year period was 10-50% less than that of healthy trees, depending on proportion of root system killed.

Twenty- to 40-year-old Norway spruces with butt rot had one-sided root distributions because *Armillaria* had killed one or more primary roots through which it had entered the stem early in the life of the tree. A reaction zone from which bacteria could be isolated extended as far as 50 cm up the stem (Yde-Andersen 1958). Butt rot of older Norway spruce was recorded by Molin and Rennerfelt (1959). In Britain, butt rot of conifers is commonly initiated when a small tap or sinker root is killed. The decay usually is limited to the lower 60 cm of the stem. Of species grown in Britain, Norway and Sitka spruces and western hemlock are most susceptible to butt rot while Douglas-fir, true firs, pines, and larches show considerable resistance (Gladman and Low 1963).

Armillaria root disease may kill its hosts by girdling the stem at the root collar. Prior to death, diseased trees may be windthrown due to decay of structural roots (Gladman and Low 1963, Shaw and Toes 1977), or they may be attractive to bark beetles which kill all or part of the tree (Cobb 1989).

Physiology of Symptom Development and Host Killing

The physiological basis of symptom development and host mortality is little understood for *Armillaria*, but two hypotheses have been proposed. First, symptoms develop as a direct result of the fungus physically disrupting the host's vascular system and the host's responses to it. Second, *Armillaria* species may produce metabolic toxins. The first hypothesis has been accepted by many investigators due to the nature of symptoms induced by *Armillaria*, particularly in the foliage. In mature conifers, shoot growth declines and the amount and color of foliage change gradually over several to many years as *Armillaria* destroys the host's vascular tissue. This view is supported by the results of Kile and others (1982), who found that patterns of electrical resistance were similar in mechanically girdled trees and those killed by *A. luteobubalina*. However, no experimental studies are known of host physiological parameters relative to location or extent of root system infection.

Several authors have postulated that symptoms are caused by a toxin produced by *Armillaria*. Orchard

trees affected by *Armillaria* appeared to exhibit symptoms of toxicity, possibly due to effects of metabolic products of the fungus (Zeller 1926). He suggested that branches died from toxic products since only branches above diseased roots showed symptoms, and pruning an infected root did not result in branch death. This view is supported by results of Thornberry and Ray (1953) who obtained a dark brown protein-like pigment from a liquid culture of *Armillaria*. The fungus had been isolated from a wilting peach tree. The substance induced wilting in tomato seedlings and peach twigs and penetrated 15-20 mm into vascular tissues. However, electrical resistance measurements around actively expanding lesions did not show that *A. luteobubalina* produces any systemic effects in eucalypts (Kile and others 1982).

Further research is needed to clarify the physiology and biochemistry of killing of host tissues (see chapters 3 and 7). Understanding this process could lead to characterization of pathogenic species and suggest what makes a host resistant to disease development.

Disease Diagnosis

Woody plants express diverse symptoms which may be categorized, in approximate chronological order, as follows: reduction of shoot growth, changes in foliage characteristics, crown dieback, stress-induced reproduction, basal stem indicators, and death. Generally, the nature of the symptoms and their rate of development relate to the position of attack and the rate of destruction of the host root system. If the disease progresses rapidly or the host is small, not all symptoms may be evident (Hartig 1874, Edgar and others 1976). Symptom development in conifers was more pronounced on vigorous hosts (Buckland 1953).

Above-Ground Symptoms on Individual Plants

Reduction of Shoot Growth

On conifer seedlings and trees up to about 10 years old, *Armillaria* rarely reduces shoot growth prior to death because killing occurs within a few months to a year after infection (Gibson 1960, Hartig 1874, Hintikka 1974). By contrast, the slower progress of the disease in older conifers causes a decline in shoot growth (fig. 5.9) which may be evident for many years (Molin and Rennerfelt 1959). In 80- to 100-year-old Douglas-fir, Bloomberg and Morrison (unpubl.) found terminal-shoot growth on diseased trees had declined for the previous 15-30 years. Actual time depended on the time since infection. Fruit trees affected by Armillaria root disease may have a stunted appearance due to a shortening of internodes (Barss 1913, Cooley 1943).

Changes in Foliage Characteristics

On conifers which are killed quickly, foliage turns red or brown as it dries. When the disease progresses slowly, as in older trees, foliage gradually becomes stunted, chlorotic, and sparse (fig. 5.9). These changes usually occur throughout the crown (Hartig 1874, Molin and Rennerfelt 1959, Morrison 1981, Williams and others 1989). Symptoms in the crowns of young Douglas-firs are frequently accompanied by prolific resin blisters on the stem and branches (Buckland 1953).

Small hardwood trees frequently are killed so rapidly by *A. tabescens* that symptoms are not evident until the foliage withers and dies (Rhoads 1956) whereas the first indication of infection on larger trees is a thin crown with small leaves (Guillaumin 1977, Sokolov 1964). Trees later show gradual yellowing and defoliation followed by rapid wilting and dying of individual

FIGURE 5.9 — A 12-year-old Douglas-fir showing reduced shoot growth (for 2 years), chlorotic foliage, and a stress-induced cone crop. (D.J. Morrison)

limbs above diseased roots (Barss 1913, Bliss 1944, Rhoads 1956). On apple trees, premature defoliation is sometimes an indicator of *Armillaria* infection (Marsh 1952); and on diseased stone fruit trees, leaves roll along the mid-rib and wilt (Cooley 1943). Attacked by *A. luteobubalina*, eucalypt saplings up to 25 years old die suddenly (fig. 5.10), showing little deterioration of crowns before death (Edgar and others 1976). On older saplings, leaves show gradual reddening followed by browning and plant death (Pearce and others 1986). In pole-size to mature eucalypts, *A. luteobubalina* causes a general reduction in leaf density, drooping of foliage, epicormic shoots along branches, and eventually a dead top (fig. 5.11). Large trees which could not compartmentalize infections usually die 2-8 years after visible crown deterioration appears (Edgar and others 1976, Pearce and others 1986).

Crown Dieback

In pole-size to mature eucalypts attacked by *A. luteobubalina*, dieback of fine twigs and branches may lead to a dead top (Edgar and others 1976). Cooley (1943) observed that limbs on apple trees ceased growth and died on the same side as the affected root. Frequently, the combined action of Armillaria root disease and other biotic or abiotic agents has been associated with crown dieback and eventual mortality of many forest species, such as those noted in chapter 7 and table 8.3.

FIGURE 5.10 — A pole-stage mountain grey gum tree killed by *A. luteobubalina*. Little crown deterioration occurred prior to the sudden death of the tree. (G.A. Kile)

FIGURE 5.11 — Messmate stringybark trees showing stages in crown decline caused by *A. luteobubalina*. (G.A. Kile)

Stress-induced Reproduction

Many woody plants respond to advanced infection by producing a seed crop, usually in the season before death. Thus, tung oil trees produce nuts which are smaller than normal (Rhoads 1956), orchard trees produce poor, stunted fruit (Barss 1913), citrus trees develop an abnormally heavy bloom (Rhoads 1948), and conifers produce cones which are smaller but may be more numerous than normal (fig. 5.9) (Buckland 1953).

Basal Stem Indicators

Woody plants attacked by *Armillaria* frequently produce exudates or develop cankers, cracks, or flutes at or just above the root collar. Genera of conifers which normally have resin canals (*Pseudotsuga*, *Picea*, *Larix*, and *Pinus*) or which form traumatic resin canals (*Tsuga* and *Abies*) may produce resin that exudes through fissures (fig. 5.12) in the bark of the root collar and lower bole (Buckland 1953, Gibson 1960, Hartig 1874, Hintikka 1974, Rykowski 1975). Usually, resin exuda-

FIGURE 5.12 — Copious basal resinosis on a radiata pine attacked by *A. novae-zelandiae* or *A. limonea*. (C.G. Shaw III)

tion is not evident above-ground until the fungus is near or has reached the root collar. Responding to advanced *A. tabescens* attack, citrus trees occasionally (Rhoads 1948) and stone fruits commonly (Rhoads 1956) produce gum in the cambial region which may be so copious as to exude through cracks in the bark. Latex exudes from rubber trees at the root collar in the last stages of the disease (Riggenbach 1966). Exudation of kino through stem and root bark occurs on some mature eucalypt trees infected by *A. luteobubalina;* and from stems of trees less than 20 years old, it may be abundant, permeating and blackening the adjacent soil (Edgar and others 1976, Kile 1981).

Infections by *Armillaria* in 20- to 70-year-old Douglas-fir, white pine, and other conifers may be arrested after killing cambium at the root collar above a diseased root. Callusing occurs around the margin of the lesion. When fresh, lesions are resinous and have mycelial fans beneath the bark. Later, after the bark sloughs, the lesions can still be recognized by their short length, broad triangular shape, and the impressions of mycelial fans on the scar face (Molnar and McMinn 1960). Conical basal scars on eucalypt stems (fig. 5.13) are frequently associated with *A. luteobubalina* infection (Kile 1981, Pearce and others 1986, Shearer and Tippett 1988). In citrus, basal lesions extend up to 35 cm above one or more diseased roots and may serve as entry points for other wood-rotting fungi (Rhoads 1948). The lesion at the base of some oil palms remains localized, dried, and apparently sealed off from the healthy tissue within; a mass of new roots forms above the canker (Wardlaw 1950). West African rubber trees infected with *Armillaria* or with *Rigidoporus lignosus* (Kl.) Imaz develop flutes at the stem base starting at the root collar near the point of infection (Riggenbach 1966).

A diagnostic symptom of Armillaria root disease on woody plants such as tea, coffee, and cacao in tropical or subtropical regions is the conspicuous longitudinal cracks that appear at the root collar and quickly extend up the trunk, hence, the name "collar crack" (Dade 1927). The cracks are longer and more numerous on the side of the tree where infection occurred. Similar cracks were observed on the roots and lower stem of citrus attacked by *A. tabescens* (Rhoads 1948) and on the roots of several hardwood species (Sokolov 1964).

In standing trees, heartwood decay (butt rot) does not produce external signs unless it is associated with a basal canker. In felled timber, butt rot caused by *Armillaria* may be recognized by characteristics of the decayed wood or confirmed by culturing. Where decay of structural roots is advanced in coniferous and broadleaved trees, they may be windthrown prior to death. This is particularly true where the tree is being sustained by adventitious roots.

Symptom Development in Relation to Extent of Colonization

The development of symptoms of Armillaria root disease in foliage and at the stem base depends upon the rate and degree of invasion of the host root system. Thus, on young (Gibson 1960, Swift 1968) or small trees (Rhoads 1956) in which the root system is invaded rapidly after infection, symptoms may appear just prior to death or only after the host is moribund. Death of radiata pine due to *A. novae-zelandiae* or *A. limonea* began 6 months after planting (MacKenzie and Shaw 1977). In 8- to 10-year-old plantations, an eastern white pine died 39 months after inoculation and a red pine infected by natural inoculum died 14 months after root examination showed it to be healthy (Patton and Riker 1959). On apricot trees, symptoms on aerial parts appeared only after the root collar was attacked or several large roots were killed (Guillaumin 1977); and on apple trees, girdling of the stem was complete 2-3 years after infection was first noted in one segment of the trunk (Marsh 1952).

Invasion of the root system of old or large trees usually occurs slowly over many years. Growth-ring studies on conifers 80 to 110 years old suggest that recently dead and severely affected trees became infected up to 50 years previously (Molin and Rennerfelt 1959). Consequently, symptoms develop gradually after a portion of the root system is colonized. Bliss (1944) found that Armillaria root disease was well established in citrus roots before any symptoms appeared in the crown. The fungus must reach the root collar before exudation of resin, gum, or kino becomes visible. More than half the root system of grand firs (mean age 50 years) had been killed by *Armillaria* with no apparent decline in tree vigor (Maloy and Gross 1963). Sokolov (1964) observed that the color and thickness of the crown and the incidence of cracks and resin flow on the lower bole were related to the proportion of first-order roots infected. In 80- to 100-year-old Douglas-firs, height growth reduction and the percentage of stem circumference showing basal resinosis were proportional to the amount of the root system colonized by *A. ostoyae* (Bloomberg and Morrison 1989). Crown symptoms on these trees were not obvious until one-half to three-quarters of the primary roots had been invaded. Crown dieback increased with increasing root collar infection in eucalypts attacked by *A. luteobubalina* (Edgar and others 1976, Kile 1981); the height of infection on stems was positively correlated with circumference infected (Kile 1981).

FIGURE 5.13 — Basal lesion on mountain grey gum caused by *A. luteobubalina*. (G.A. Kile)

Confirmation of *Armillaria* Occurrence

Many symptoms described above are non-specific; that is, they may be induced by a number of biotic and abiotic factors. To confirm Armillaria root disease, the root collar and lower bole of the tree must be examined for signs specific to the fungus. Those signs include mycelial fans, rhizomorphs, basidiomes, and decay. *Armillaria* may also be confirmed by culturing from the host. Many of the signs are useful for identifying stumps and roots which are within disease centers or on cutover sites, and which may be inoculum sources for the next rotation.

Mycelial Fans

On plants showing symptoms of advanced infection and on those recently killed, creamy-white mycelial sheets up to 10 mm thick occur in the cambial zone of roots and the lower bole (Greig and Strouts 1983, Morrison 1981, Williams and others 1989). The mycelial sheets, commonly known as fans and occasionally referred to as xylostroma, are the most useful diagnostic characteristic of *Armillaria* species in woody plants (figs. 5.14A,B). The mycelial fans of some *Armillaria* species are marked with perforations (fig. 5.15) 0.2-3 mm in diameter (Gibson and Corbett 1964, Kile and Old 1982, Rhoads 1945). In plants which have been dead for several years, mycelial fans usually can be found in roots below-ground but have disappeared from above-ground parts due to competing fungi or to unfavorable environmental conditions, such as desiccation. On conifers, impressions of fans in resin and bark may be present for several years after fans disappear (fig. 5.16).

Several reports of *Armillaria* on African crops (Dade 1927) and on hosts of *A. tabescens* in Florida (Rhoads 1948) refer to frills of xylostroma, at first cream-colored then becoming dark brown with age, which protrude from the longitudinal fissures in the bark. The description by Dade (1927) indicates that xylostroma sheets are extensions of subcortical mycelial fans which become melanized when exposed to air, an observation confirmed by Rhoads (1948).

Rhizomorphs

Rhizomorphs are initiated on the food base from the edges of mycelial fans, either subcortically when conditions such as loosening of bark prevents further growth

A B

FIGURE 5.14 — A: Mycelial fans of *A. ostoyae* in the cambial zone of an 8-year-old Douglas-fir. (D.J. Morrison). B: Mycelial fans of *A. luteobubalina* on brown barrell eucalypt. Note rhizomorphs emerging from the fan margin where the bark was loosened. (G.A. Kile)

FIGURE 5.15 — Perforated mycelial fans of *A. luteobubalina* developed in vitro in stem segments of silver wattle. (G.A. Kile)

of the fan (fig. 5.14), or into soil when the fan reaches the bark-soil interface (Morrison 1972). For up to 1 cm from the growing tip, a rhizomorph is white; with increasing distance from the tip it becomes red-brown, brown, and finally black. A rhizomorph is hollow near the growing tip; however, within 2 cm, the hollow becomes filled with randomly arranged fiber hyphae in a mucilaginous matrix (Redfern 1973, Schmid and Liese 1970). Rhizomorphs in soil and on the surface of roots are usually 1-3 mm in diameter (Morrison 1972, Pearce and others 1986, Redfern 1973). Occasionally, rhizomorphs in soil, probably of *A. gallica*, are 5 mm in diameter (Redfern 1973). Rhizomorph structure is discussed fully in chapter 3.

In the north temperate zone (Greig and Strouts 1983, Wargo and Shaw 1985), New Zealand (Hood and Sandberg 1987), and at higher elevations in East Africa (Gibson 1960), India (Satyanarayana and others 1982) and Sri Lanka (Gadd 1930), rhizomorphs of *Armillaria* species grow freely through soil and on the surface of roots. The rate of growth and distance from the food base that they will grow varies greatly among species. Species with monopodially branched rhizomorphs, such as *A. gallica*, often produce extensive networks in

FIGURE 5.16 — Impressions of *A. ostoyae* mycelial fans on the inner bark of Douglas-fir. (D.J. Morrison)

soil, whereas dichotomously branched species, notably *A. mellea* (Rishbeth 1982), appear to be restricted to within a few centimeters of the food base. For this reason, the usefulness of rhizomorphs as a diagnostic feature is limited, particularly at the specific level.

At low elevations in the tropics, rhizomorphs are not found in soil or on roots (Dade 1927, Gibson 1960, Rishbeth 1980, Swift 1968), although occasionally they grow up to 2 cm into soil and then die (Dade 1927). In Australia, rhizomorphs either were not observed in the field (Kile 1981, Shearer and Tippett 1988) or were found only on the surface of roots (Kile 1980b, Pearce and others 1986).

Basidiomes

Basidiomes occur in clusters arising from mycelial fans in the host or in small numbers from rhizomorphs on the host or in soil. Basidiomes facilitate surveys of disease incidence (Pearce and others 1986) and identification of the *Armillaria* species (see chapter 1). Basidiomes often occur on or near hosts lacking other signs and symptoms. In temperate regions, fruiting occurs from mid-summer to mid-winter, depending on latitude and weather. Precipitation and favorable temperatures are required to initiate fruiting and for basidiome development. Basidiomes which develop slowly due to cold or dry weather may have short, thickened stipes and small thick pilei; weather may also affect basidiome color (Kile and Watling 1981). In tropical regions, basidiome formation varies from rare in Sri Lanka (Gadd 1930) and East and Central Africa (Wallace 1935, Gibson 1960b, Swift 1972) to common in West Africa (Dade 1927, Riggenbach 1966), where it occurs almost exclusively in the wet season (Wardlaw 1950).

Decay

Armillaria species cause a white rot of woody tissues as lignin and cellulose both decompose. The appearance of decayed wood varies somewhat among hosts. In conifers, wood with incipient decay is stained gray to brown, often with a water-soaked appearance. Later, decayed wood becomes yellow-brown and stringy (figs. 5.17 and 5.18) and is finally reduced to a very wet, stringy rot with pale yellow flecks (Greig and Strouts 1983, Williams and others 1989). Decayed wood of broadleaved hosts is watersoaked and white to yellow, becoming spongy and ultimately distinctly gelatinous (Greig and Strouts 1983, Rhoads 1956).

Pseudosclerotial plates (zone lines) are common in woody tissues decayed by *Armillaria* species (Campbell 1934, Lopez-Real 1975, Greig and Strouts 1983, Podger and others 1978). These plates are composed of pigmented bladder hyphae which are identical with the cells comprising the outer coat (rind) of mature rhizomorphs (Campbell 1934, Lopez-Real 1975). Wood decayed by some, but not all, *Armillaria* species is bioluminescent (Kile 1980b, Podger and others 1978). The biochemistry of bioluminescence is discussed in chapter 3.

Isolation Technique and Appearance in Culture

The presence of an *Armillaria* species in host tissue may be confirmed by culturing colonized bark or wood or subcortical mycelium on a medium such as potato dextrose or malt extract agar. Molds or bacteria may be suppressed by acidifying the medium or amending it with a fungicide such as o-phenylphenol (Russell 1956) or benomyl (Hunt and Cobb 1971, Maloy 1974). Isolation of *Armillaria* from root tissues of dead and dying trees increased by 40% on malt agar amended with o-phenylphenol (Whitney and others 1978). The selective media developed by Kuhlman (1966) and Kuhlman and Hendrix (1962) for isolating *H. annosum* from wood and its spores from soil also is selective for *Armillaria* (Shaw 1981a). The fungus may be isolated from rhizomorphs by first washing short lengths in water then soaking them in 10% hypochlorite for 5 min (Rishbeth 1978b). Hood and Sandberg (1987) made isolations from rhizomorphs after dipping them in 95% ethanol, surface sterilizing in 10% hydrogen peroxide, and washing in distilled water.

Nobles (1948) suggested that *Armillaria* cultures are recognizable from macroscopic appearance alone, their red-brown crustose areas, rhizomorphs, and frequent luminosity of young, actively growing colonies being unique. Her description is based on four isolates, three of which were from conifers in British Columbia and

FIGURE 5.17 — Yellow stringy decay of Douglas-fir root wood caused by *A. ostoyae*. (D.J. Morrison)

FIGURE 5.18 — *Armillaria*-caused butt rot of Norway spruce. (B.J.W. Greig)

Washington. It is likely that the description is based on cultures of *A. ostoyae*. However, these features are characteristic of most, if not all, species of *Armillaria*. Differentiation of vegetative isolates of *Armillaria* is discussed in chapters 1 and 2.

Biotic and Abiotic Conditions Causing Similar Symptoms

Any agent or condition which affects the root system of a woody plant may cause some or all of the symptoms described above. In conifers, root diseases caused by *H. annosum* (Greig and Redfern 1974), *Phellinus weirii* (Murr.) Gilbn. (Thies 1984, Wallis 1976), *Inonotus tomentosus* (Fr.) Teng (Whitney 1978a) and *Leptographium* spp. (Wingfield and others 1988) may cause crown symptoms similar to those of *Armillaria*. On apple trees, winter injury to the roots or root collar or root suffocation due to flooding can induce symptoms similar to Armillaria root disease (Cooley 1943). Stem girdling or root killing due to any cause induces foliage symptoms in citrus similar to those of Armillaria root disease (Rhoads 1948).

Disease Detection

Detecting Armillaria root disease in production forests, amenity woodlands, and agricultural plantations depends on observable symptoms in the crown and on the stem base plus signs of the fungus such as mycelial fans, rhizomorphs, and basidiomes on the host. Diseased trees occur as scattered individuals or in centers which reflect the distribution of the *Armillaria* species. Characteristics of disease centers are discussed in chapters 8, 9, and 10.

Aerial photography and ground surveys conducted independently or in combination have been used to detect root diseases, including those caused by *Armillaria*. Choice of survey method is influenced by the purpose of the survey. For example, the survey may intend to determine presence or absence of root disease, estimate wood volume in diseased trees, delineate distribution of disease, or provide input data for modeling purposes (see chapter 10). Aerial photographs (Kable 1974) and stem maps (Marsh 1952) also have been used to detect and record progress of Armillaria root disease in agricultural plantations.

Using aerial photography permits large areas of forest to be inspected rapidly for visibly affected trees, for quantifying effects, and for providing a record of disease occurrence. Some ground inspection is required to identify the pathogenic species involved and to verify the photographic assessment. The choices of image scale and film emulsion to be used are based on stand structure, ease of defining disease signature, and purpose of the imagery. While detection of disease centers and affected single trees may be accomplished at scales up to 1:10000 (Gregg and others 1978, Murtha 1972, Myers and others 1983, Williams 1973), larger scale imagery, 1:1000-1:2000, may be necessary to provide rea-

sonable accuracy in delineating areas affected. Generally, relatively large scale imagery, 1:3000-1:6000, is most often used for detecting and quantifying individual trees or centers (Gregg and others 1978, Myers and others 1983, Wallis and Lee 1984, Williams 1973, Williams and Leaphart 1978). Color and false color (color infrared) emulsions are frequently used (Gregg and others 1978, Heller and Bega 1973, Williams and Leaphart 1978); black and white may also be effective (Johnson and Wear 1975).

In western North America, the signature of root disease centers on aerial photographs included openings in the forest canopy with dead or nearly dead standing trees on the margins, snags, and windthrown conifers, and generally a shrub cover and some young trees in the opening (Wallis and Lee 1984, Williams and Leaphart 1978). Dead trees and crown decline characterized *A. luteobubalina* centers on photographs of jarrah forests in Western Australia (Shearer and Tippett 1988).

Ground evaluations using various survey procedures are efficient if areas are small or if precise disease location and damage measurements are required. Survey design varies from regularly or randomly spaced transects to systematically spaced variable and fixed-radius plots (Jacobi and others 1981). Pearce and others (1986) used random reconnaissance, transect and plot surveys to determine the occurrence of basidiomes and the incidence of infection in stumps, saplings, and trees. The ground survey method developed for *P. weirii* (Bloomberg and others 1980a,b) and modifications for multiple-disease recording and stratification by infection intensity (Bloomberg 1983) are applicable to surveys for Armillaria root disease. This transect sampling system involves randomly located sets (grids) of lines to estimate the incidence, distribution, and area of root disease. Estimates of diseased area are derived from length of transect intersecting root disease centers and probability of occurrence. Random location of gridlines in a stand results in independent estimates for each grid, hence the variance of their means can be estimated.

The Bloomberg method is difficult to apply in logged, burned, or open stands with diffuse disease distribution because locating infection boundaries can be difficult. For that reason, Kellas and others (1987) used systematically located transects with variable-sized plots around selected stumps to assess infection by *A. luteobubalina* in regeneration, regrowth, and overwood trees. Incidence and severity of Armillaria root disease can be assessed during inventory surveys (B. Geils, unpubl.). Ground survey data such as that frequently collected by the USDA Forest Service (1986) may be used to initialize a model of Armillaria root disease (see chapter 10), if augmented to include stumps infected with root disease (Stage and others 1990).

Where survey information is required for large areas, multi-stage or double sampling designs incorporating aerial photography and ground evaluations can be employed (Stewart and others 1982, Williams and Leaphart 1978, Wood 1983).

Conclusions

The infection process has been observed on hardwood and coniferous hosts. Post-infection disease development has been observed for a few host species but not throughout a rotation. The response to infection by a variety of host species has been recorded, primarily at the macroscopic level, but less is known of the interactions between hosts and *Armillaria* at the biochemical level. The effects of Armillaria root diseases on their hosts, growth loss, decay, and mortality, are known. Symptoms of Armillaria root diseases which are nonspecific include reduction of shoot growth, changes in foliage characteristics, crown dieback, stress-induced reproduction, basal stem indicators, and death. Signs specific to *Armillaria* species are subcortical mycelial fans, rhizomorphs, and basidiomes. Cultures of *Armillaria* have distinctive characteristics. Ground and aerial methods for detecting Armillaria root diseases and ground procedures for determining disease area have been developed although work is needed to improve their utility. Understanding the biochemistry and physiology of the host-parasite interaction and studies of disease development during a rotation for representative combinations of host and *Armillaria* species remain the most urgent research needs relating to infection and disease development.

CHAPTER 6

Pathogenicity and Virulence

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The terms pathogenicity and virulence both refer to an ability to cause disease. That *"Armillaria mellea"* can cause disease has been known for over a century, but its propensity to do so has been a matter of controversy. Rhizomorphs commonly surround tree roots without infecting them, yet *Armillaria* may cause extensive mortality elsewhere in the same area. Such observations were interpreted by some early authors as indicating that trees in affected areas were weakened or predisposed to infection in some way (Day 1927b, 1929). Others, for example Piper and Fletcher (1903) and Childs and Zeller (1929), proposed that there were several forms of the pathogen that differed in virulence.

According to the former view, *Armillaria* was a secondary pathogen capable of attacking only trees with lowered resistance. Thus, Day (1929) concluded that "all the evidence goes to show that it is always secondary to some other factor acting as a primary cause of disease." Boyce (1961) stated that the fungus "does not attack thrifty trees" and Gremmen (1976) expressed the view that "control of *A. mellea* in forestry ... has no effect since the fungus is not the primary cause of dieback." Contrary to these assertions, however, there are early accounts of *Armillaria* disease (for example Hendrickson 1925, Zeller 1926) that describe attacks by a fungus with every appearance of a "virulent primary pathogen," as it was termed by Patton and Riker (1959). Dade (1927) similarly described the behavior of *"Armillaria mellea"* in tropical West Africa, and Brooks (1928) regarded it as "perhaps the most dangerous subterranean parasite of trees, bushes and certain herbaceous plants."

Many contradictions regarding the pathogenic behavior of *"Armillaria mellea"* can now be understood as having arisen from observations made on different *Armillaria* species. They can differ markedly in pathogenicity yet closely resemble each other in the appearance of their basidiomes, rhizomorphs, and mycelial sheets. The extremely low pathogenicity of some species may partly explain the dismissive attitude some earlier authors held toward *Armillaria* as a pathogen.

Pathogenicity, Virulence, and Disease

Distinguishing between pathogenicity and virulence is especially important when so many species and so many different hosts are involved. "Pathogenicity" means the quality or characteristic of being able to cause disease as applied to a genus or species (British Federation of Plant Pathologists 1973). "Virulence" means the observed infective capacity of individual entities of a pathogenic species (British Federation of Plant Pathologists 1973).

Pathogenicity of an *Armillaria* species was first established in an inoculation experiment by Hartig (1874) though his method fell short of satisfying Koch's postulates, which are now generally accepted as the requirements for proving pathogenicity (British Federation of Plant Pathologists 1973). An extensive world literature on *Armillaria* now contains enough data from inoculation experiments to leave no doubt that several pathogens occur in the genus.

Some physiological attributes of the fungus that may be associated with high or low virulence are discussed in chapter 3, but the genetic basis of virulence in *Armillaria* is largely unknown. Two studies have shown that haploid isolates derived from single basidiospores may display high virulence, in some cases as high as the parent isolate (Raabe 1972, Shaw and Loopstra 1988). The wider genetic significance of this finding and its relevance to field behavior remain to be investigated. Reaves and others (1988) suggested that the occurrence of virus-like particles in the cytoplasm of some *Armillaria* isolates might be associated with high virulence, but little evidence supports this hypothesis.

Saprophytic and Parasitic Behavior

Armillaria species have both saprophytic and parasitic phases in their life cycle, but distinguishing the two may be difficult in an activity such as colonization of a moribund stump. By causing root- and butt-rot in standing trees, *Armillaria* species can also be classified as perthophytes because they utilize dead tissues in living hosts (British Federation of Plant Pathologists 1973). Most of the methods of capturing resources for saprophytic or perthophytic exploitation that have been outlined in chapter 4 depend on the fungus' abilities as a parasite even though the tissues involved might be of extremely low vigor, as in stumps and dying trees.

Pathologists and mycologists now recognize that *Armillaria* species differ markedly in pathogenicity. Highly pathogenic species survive saprophytically in the hosts they kill through their parasitic activities, whereas weak pathogens probably exist for the most part as saprophytes or possibly perthophytes (Korhonen 1978, Rishbeth 1985a, Wargo and Shaw 1985). This diversity poses the question whether weakly pathogenic species are better able than highly pathogenic species to colonize moribund tissues and compete with saprophytic micro-organisms. Little information is available on this subject, and it is clearly an area that merits further research.

Rishbeth's (1985a,b) experiments with excised root and stem material demonstrated that, in some circumstances at least, the weak pathogen *A. gallica* is no more capable than the highly pathogenic *A. mellea* of colonizing woody material with residual host resistance and may even have an inferior ability to penetrate intact bark on such material. The same studies suggest that these two species may differ little in their ability to colonize completely dead material, and both may possess considerable competitive saprophytic ability (*sensu* Garrett 1956a, 1970).

Armillaria ostoyae, another highly pathogenic species, did not perform as well as *A. mellea* and *A. gallica* in Rishbeth's (1985a,b) tests with excised material. In western North America *A. ostoyae* is considered incapable of colonizing stumps that were not already infected as living trees (Filip 1989a). Although it is one of the assumptions underlying recent models of disease development (see chapter 10; McNamee and others 1989), the reasons for this apparent inability are not clear. It may reflect the species' limited capacity for spreading by rhizomorphs as much as any deficiency as a saprophytic competitor.

An important attribute of weakly pathogenic species is an ability to act as facultative parasites on stressed or sickly hosts (Kile 1980b, Rishbeth 1985a, Wargo and Shaw 1985). However, many observations suggest that highly pathogenic species are also capable of invading weakened hosts (Davidson and Rishbeth 1988, Dumas 1988, Gregory 1989, Guillaumin and others 1989a, Rishbeth 1985a). In nature, it is probable that the weakly pathogenic species more often do so (Kile 1980b, Kile and Watling 1983, Rishbeth 1982).

Quite possibly, some of the less pathogenic *Armillaria* species have evolved strategies, such as rhizomorph behavior, that confer advantage of position in exploitative situations (Gregory 1985, Rishbeth 1985a, Wargo 1984b, see chapter 4). Indeed, the paucity of data permits more general speculation that the undoubted success of such species owes less to any greater ability to penetrate and invade weakened or dead tissues than to an ecology that affords them the maximum opportunity to encounter such material. This is more fully discussed in other chapters, but it is relevant to note here that such considerations necessitate great caution in interpreting observations and experiments on pathogenic behavior.

Conditions For Disease

Implicit in the definition of pathogenicity is the qualification that measurement should be made under specified conditions. Among the most important elements that may influence the expression of pathogenicity are the host, the external environment, and the nutrition of the pathogen. Pathogen nutrition is contained in the concept of inoculum potential which was elaborated by Garrett (1956a, 1970). The ability of a pathogen, whatever its inherent virulence, to cause disease is strongly influenced by the energy available to it at the host surface. The subject of inoculum potential is discussed in chapter 4.

Host resistance is an important constraint on disease, and many studies have shown that susceptibility to *Armillaria* disease differs among host species. European forest hardwoods have been shown to possess considerably more resistance than native or exotic conifers (Redfern 1978, Rishbeth 1984), results that are in accord with most field observations. However, some conifers are notably resistant (Guillaumin and Pierson 1978) and some hardwood genera, *Prunus* and *Citrus*, for example, are notoriously susceptible (Guillaumin and Pierson 1978, Raabe 1967, Wilbur and others 1972). Differences in susceptibility of woody species within individual genera have frequently been demonstrated (Benjamin and Newhook 1984b, Guillaumin and others 1989b, Proffer and others 1988); and Azevedo (1970-71) found that two forms of the same species (Japanese redcedar) also differed.

Host resistance is not only a genetic attribute but also a result of circumstances. Notwithstanding the historic controversy over the role of host predisposition in *Armillaria* pathogenesis, factors associated with low host resistance will favor disease. Good circumstantial evidence from several parts of the world indicates young trees are more prone to infection than older trees of the same species (Gibson 1960, Ono 1970, Pearce and others 1986, Redfern 1978), and many pathologists believe stress imposed by ill-health, injury, or unsuitable growing conditions can increase susceptibility (see chapter 7).

The best known limitations imposed by the external environment on the activities of pathogenic *Armillaria* species are the effects of soil on rhizomorph growth and production. The complicated relationships between rhizomorphs and disease are discussed briefly in the following sections and more fully in chapter 4.

Decay and Disease

The commonly accepted definitions of disease refer to deviation from normal functioning of physiological processes (British Federation of Plant Pathologists 1973). It is therefore arguable whether butt rot, which involves the decay of largely non-living interior wood in living trees, constitutes disease. We will accept it as such since living cells in the wood are likely to be affected to some degree in many cases. The ability of *Armillaria* species to cause decay in standing trees is therefore an expression of pathogenicity though it appears not to have been investigated experimentally. Most experiments assess virulence entirely by the effects of the pathogen's development in the phloem and cambium.

In practice, root killing and root decay are often not clearly separable since one closely follows the other. Nevertheless, these processes involve the capacity to invade and exploit two quite different tissues, and the decay-causing ability of an isolate is not necessarily related to its capability as an agent of tree mortality. Decay has been little studied in *Armillaria*, but field observations in Europe (Gregory 1989, Korhonen 1978, Rishbeth 1982) suggest that species with limited ability to kill trees are associated with butt rot at least as often as highly pathogenic species.

Host Specialization

Many *Armillaria* species have a wide host range, both among the genera which occur naturally in their habitat and among exotics. For example, the Australian species *A. luteobubalina* not only attacks many native tree and shrub species in many genera but is also highly pathogenic to some North American conifers (see chapter 8; Morrison 1989). Such behavior does not preclude the existence of adaptive relationships between particular pathogens and particular hosts ("host specialization" or "host preference"), though few have been clearly demonstrated in *Armillaria*. In Europe, the area from which most data are available, *A. ostoyae* appears to be better adapted to coniferous hosts and *A. mellea* to hardwoods (Guillaumin and others 1985; Guillaumin and Lung 1985; Guillaumin and others 1989a; Rishbeth 1985a; Siepmann 1985). However, distinguishing the effects of host specialization from those of site history and pathogen ecology is often difficult. Both may limit the opportunities for contact between the fungus and some potential hosts.

Assessing Pathogenicity and Virulence

The ability to cause disease can be estimated from direct measurement of the amount of disease actually caused in inoculation trials, observation of field behavior, or an assay of some feature thought to be associated with the pathogen's ability to cause disease. All three approaches have been attempted with *Armillaria*, but the first two have undoubtedly been the most useful.

As already discussed, the intrinsic ability of an *Armillaria* species to cause disease may be modified by circumstances and environment. Hence, inoculation trials must be conducted under specified conditions, choice of which is exceptionally difficult with tree-root pathogens, such as *Armillaria*, that have a wide host range and that can attack trees of virtually any age. Moreover, the infection of such a massive and well-protected structure as a woody root requires a specialized pathogen (*sensu* Garrett 1970) for which the method of infection, and particularly the necessary inoculum potential, may be difficult to achieve artificially. For many *Armillaria* species, the chief means of infection is the rhizomorph, a specialized structure that may develop only under certain conditions and the efficacy of which is governed partly by the energy supplied to the infective tip (Garrett 1956b).

Choice of Host for Inoculation Trials

Most investigators have selected trees or shrubs for pathogenicity trials. However, some have attempted to avoid the considerable difficulties of experimentation with intact woody hosts by using parts of plants or plant organs which may possess much less host resistance than a tree but might retain enough to repel isolates of low virulence.

Large vegetable roots and tubers have proved valuable subjects for the study of infection biology. Garrett (1956b), Thomas (1934), and van Vloten (1936) used

potato tubers to demonstrate apparent differences in virulence between *Armillaria* isolates. Gregory (1984, 1985) and Rishbeth (1984) also attempted to use potato tubers to test virulence, comparing the results obtained with them to those obtained by using the same isolates on young trees. Although Gregory (1984, 1985) found some correspondence, infection of the tubers generally occurred too readily for it to be pursued as a useful discriminatory method.

The dangers of using material with low host resistance for determining the virulence of *Armillaria* isolates may be increased when the "host" is an excised root or stem. The ability to colonize moribund material may be of equal evolutionary advantage, and hence as well developed, in pathogens of low virulence as in those of high virulence. As discussed by Rishbeth (1985a,b), there is compelling evidence that *Armillaria* species of low pathogenicity can successfully colonize such material both in nature and in the laboratory. Indeed, the commonly used method of preparing inocula developed by Redfern (1970, 1975) depends on this very ability. Rishbeth (1984) compared the ability of several isolates to colonize excised stems and roots. His results did not encourage the use of this ability as a measure of virulence since isolates of *A. gallica* generally performed better than those of *A. ostoyae*, a reversal of the normal ranking for pathogenicity.

Among workers who have used trees or shrubs for pathogenicity tests, many have chosen to include more than one species because of known or suspected differences in susceptibility among potential hosts (Benjamin and Newhook 1984b, Guillaumin and Lung 1985, Guillaumin and Pierson 1978, Kile 1980b, Morrison 1982b, Mugala and others 1989, Proffer and others 1988, Raabe 1967, Rishbeth 1985b, Shaw and Loopstra 1988). Other investigators have confined themselves to a host in which *Armillaria* is a current economic problem (Leach 1937, Mallett and Hiratsuka 1988, Ono 1970, Podger and others 1978, Wilbur and others 1972). The type, age, and method of cultivating experimental subjects have differed greatly, but four plant types have been commonly used: very young seedlings grown under laboratory conditions, potted plants, plants in field plots, and established trees.

Several attempts have been made to use seedlings under sterile or near-sterile conditions for laboratory infection studies (Christensen 1938, Irvine and McNabb 1962, Rayner 1930, Rishbeth 1984, Zollfrank and Hock 1987). In these experiments, infection either hardly occurred at all (Christensen 1938, Rayner 1930, Rishbeth 1984) or was achieved only by growing the seedlings in a culture medium permeated by the fungus (Irvine and McNabb 1962, Zollfrank and Hock 1987). The symptoms reported in some cases do not resemble those that occur in the field (Rayner 1930, Zollfrank and Hock 1987). Laboratory methods inevitably limit host size and the type of inoculum that can be used, so results must be considered as bearing little relationship to pathogenesis in vivo.

Inoculating young trees in containers (figs. 6.1, 6.2) has, by contrast, provided much valuable information on the infection biology and pathogenicity of *Armillaria*. In North America, this method contributed to several important studies of *"Armillaria mellea"* (Bliss 1946; Patton and Riker 1959; Raabe 1955, 1967, 1972; Shaw 1977; Thomas 1934), and it formed the basis of several recent investigations into the pathogenicity of the currently recognized North American species (Mallett and Hiratsuka 1988, Morrison 1989, Mugala and others 1989, Proffer and others 1988, Shaw and Loopstra 1988). European, Asian, and Australasian studies have also made extensive use of container plants (Gregory 1985; Guillaumin and Lung 1985; Guillaumin and Rykowski 1980; Kile 1980b, 1981; Ono 1970; Pearce and others 1986; Podger and others 1978; Redfern 1978; Shaw and others 1980, 1981; Siepmann and Leibiger 1989). Several workers (Morrison 1989; Ono 1970; Redfern 1975, 1978; Pearce and others 1986; Proffer and others 1988) have used several plants per container with each container being treated as a plot.

Experimental field plots established in open ground have also been used effectively in *Armillaria* research. Most experimental data on virulence of European isolates derive from the field plot inoculations of Rishbeth

Figure 6.1 — Inoculation of a ponderosa pine seedling with a branch segment of red alder containing *A. ostoyae* (see Shaw 1975, 1977). The jar contains inoculum segments on which *A. ostoyae* mycelium is visible as white tufts. (G. M. Filip)

Figure 6.2 — Two treatments from Redfern's (1975) trial, photographed 18 months after inoculation with European isolates of *A. gallica* (S3) and *A. mellea* (S4) in root segments of planetree. Each container originally held 25 young Sitka spruce. *A. mellea* (S4) killed all but a few plants in this replicate (treatment total of 61%), whereas *A. gallica* (S3) killed none (less than 5% over the whole experiment). (D. B. Redfern)

(1982, 1984, 1985a,b) who primarily used 2-year-old Scots pine but also worked with other conifers and a range of hardwood trees and shrubs. Guillaumin and Pierson (1978) used 4- to 5-year-old specimens of peach, Persian walnut, downy oak, and silver fir in field trials in France. In the United States, Wilbur and others (1972) used field plots of peach. One of the few inoculation trials to have been reported for an African *Armillaria* isolate was conducted in a field plot of common tea seedlings by Leach (1937).

Relatively few inoculations of established plantation or forest trees have been reported though the hosts for the earliest recorded inoculation were 8-year-old pines in Germany (Hartig 1874). One of the first demonstrations that *"Armillaria mellea"* exhibited differences in virulence was achieved by inoculating young plantation pines in the United States (Patton and Riker 1959). Also in the United States, there has been a history of field inoculations in research on *A. tabescens* (Plakidas 1941, Rhoads 1956, Weaver 1974). The pathogenicity of two other species has been proven by field inoculation. Kile (1981) inoculated young eucalypts with *A. luteobubalina* in Australia, and Dadant (1963a) inoculated field-grown albizia with *A. fuscipes* in Madagascar. Large woodland trees have been inoculated in several other studies in which the objective was investigation of host-parasite relationships rather than straightforward testing of pathogenicity (Davidson and Rishbeth 1988, Redfern 1978, Wargo and Houston 1974, Whitney and others 1989b).

Inoculating forest or plantation trees could yield data more relevant to field experience than any other method discussed in this section. However, the practical difficulties involved are often formidable. Using containers offers ease of handling, flexibility of experimental design, and greater freedom in environmental control, but conditions in containers, even those as large as Ono (1970) and Redfern (1975, 1978) used, can be quite artificial, particularly the rooting environment. Any stress imposed by such conditions could lower host resistance and might thereby obscure differences in virulence between isolates. As noted elsewhere in this chapter, some species of *Armillaria* with limited ability as primary pathogens can nevertheless act as effective secondary pathogens on weakened trees. Conditions in containers, such as extremes of soil moisture, also may adversely affect the fungus (Guillaumin and Leprince 1979).

Growing conditions in field plots are clearly more natural than those in containers though trees are not necessarily stress-free. Morrison (1982b) mentioned drought stress as a possible factor contributing to high infection in plots established on a sandy soil. Conversely, one cannot assume that artificial or unnatural conditions are always detrimental to the host. Well-tended plants in pots or field plots may be less prone to stress, and hence potentially more resistant, than trees of the same age in some natural situations.

Container plants are usually young and are therefore likely to be less resistant to infection and killing than older trees, an obvious drawback to applying results to the field. In most pot trials, experimental plants have been seedlings, cuttings, or transplants 1-4 years old at inoculation. Exceptionally, seedlings only a few weeks old have been used (Entry and others 1986) but results in such cases are likely to have little relevance to field behavior. Field plots offer greater opportunity for using older plants, though in many such studies age at inoculation has been 5 years or less (Guillaumin and Pierson 1978; Morrison 1982b; Ono 1970; Rishbeth 1982, 1984; Rykowski 1984).

Rishbeth (unpubl.) used a range of isolates and *Armillaria* species to inoculate, in parallel trials, 1-year-old plants in pots, 2-year-old plants in field plots, and 7-year-old plantation trees of Corsican pine. Although no true comparison was possible, the data suggest that isolates of low virulence could receive higher ranking from the results of trials with young potted plants than would be justified by other methods, including field observation. Results presented by Proffer and others (1988) are also of interest in this connection. They found uniformly high mortality in cherry *(Prunus)*

seedlings inoculated with one of three *Armillaria* species including *A. gallica*, which is normally regarded as an extremely weak pathogen. Quite possibly the isolate used was of exceptionally high virulence, but more likely, the methodology gave a spuriously high result. The hosts were 1-year-old seedlings to which inocula were attached at the time of planting. As the trial ran for only 1 year, both stress of transplantation and the young age of the plants might have increased susceptibility. The large amount of inoculum used per plant was another possible factor identified by the authors.

Choice of Inoculum

Garrett's (1956a) development of the concept of inoculum potential was founded on the recognition that failure to achieve experimental infection with root pathogens was often the result of using unsuitable inocula. As discussed in chapter 4, the inoculum potential for *Armillaria* pathogenesis in vivo is almost exclusively derived from woody substrates. Accordingly, although successful inoculations of young trees have been achieved with other material, the main experimental contributions to our knowledge of *Armillaria* pathogenicity and virulence have been based on the use of woody inocula.

Some workers have used naturally infected roots (Kile 1980b, 1981; Leach 1937; Ono 1970; Proffer and others 1988; Rhoads 1938), but these are of limited value for comparative work because of the uncertainty that uniform colonization has been achieved by a single isolate. Most investigators, including some of the earliest to conduct successful inoculation experiments, have used sterilized wood pieces inoculated with pure cultures of the isolates under investigation (Bliss 1946, Guillaumin 1977, Patton and Riker 1959, Podger and others 1978, Raabe 1955, Rishbeth 1984, Shaw 1977, Thomas 1934, van Vloten 1936, Wilbur and others 1972). Some have used inocula prepared in this way to infect live stem or root pieces which have then been used to inoculate the experimental plants (Gregory 1985; Redfern 1970, 1975, 1978; Rishbeth 1972b, 1982; Siepmann and Leibiger 1989). This two-stage method has proved advantageous with some isolates that do not readily produce rhizomorphs from sterilized wood (Redfern 1970). Both methods are time-consuming because inocula take many weeks to become completely colonized, the stage at which they are usually used (Podger and others 1978, Redfern 1975, Shaw 1977). Wilbur and others (1972) incubated inocula for as long as 20 months before use. The consequences of using inocula too early have been noted by Benjamin and Newhook (1984b) who found that incompletely colonized inocula did not produce rhizomorphs and that the colonization rate varied greatly among the several types of wood that they tried. Rhizomorph production can be an important factor in achieving artificial infection, as will be discussed below, and it may be influenced directly by the food base used (Azevedo 1970-71; Morrison 1972; Redfern 1970; Rishbeth 1972b; Rykowski 1981c, 1984).

Redfern (1975) demonstrated that food base type can affect the amount of experimentally induced disease independently of how it affects the number of rhizomorphs. Choice of wood species for inocula is therefore potentially important for experimentation though the criteria used have rarely been stated. Several authors have used standard hardwood inocula for a range of hosts (Guillaumin and Lung 1985, Guillaumin and Pierson 1978, Raabe 1967, Rishbeth 1984, Shaw 1977, Shaw and others 1981, Siepmann and Leibiger 1989); Pearce and others (1986) used two different types for each host. Other workers matched inoculum to host (Dadant 1963a, Ono 1970, Podger and others 1978, Proffer and others 1988), or used unrelated species that are a common source of infection in nature (Leach 1937), or material that can be conveniently collected (Mallett and Hiratsuka 1988). The popularity of hardwood inocula even for coniferous hosts may well reflect the widespread view that hardwoods offer a superior food base for *Armillaria* species (see chapter 4; Redfern 1970, 1975).

The relative merits of using root, branch, or stem wood for inocula have received little attention although the origin could conceivably affect the fungus' pathogenic behavior. Several workers have used root segments (Dadant 1963a, Ono 1970, Patton and Riker 1959, Redfern 1975, Weaver 1974, Wilbur and others 1972), presumably reflecting the most common inoculum source in nature, but many others have achieved worthwhile results with stem or branch material (Gregory 1985, Guillaumin and Pierson 1978, Kile 1981, Morrison 1982b, Raabe 1967, Rishbeth 1982, Rykowski 1984, Shaw 1977).

The size of inocula and their positioning relative to the host have been little discussed despite Garrett's (1956b) early demonstration that both factors affect the ability of rhizomorphs to cause infection. Harrington and others (1989) and Patton and Riker (1959) attributed disappointing results in their field inoculations to under-sized inocula. Size influenced infection in Azevedo's (1970-71) and Rykowski's (1981c, 1984) experiments with young trees, but the latter still achieved infection of 3-year-old pines with inocula less than 5 cm^3. Gregory (1985) and Guillaumin and Pierson (1978) conducted successful pathogenicity trials with comparatively small inocula (1.5-2 cm diam x 4-5 cm long) used singly and placed close to the collar or major roots of the host. Other workers have generally used larger inoculum segments and several have used more than one per host. Redfern (1975) used five large segments

(2.5-5.5 cm diam x 10 cm long) in each tub (30 cm diam) of 25 small conifers, whereas Davidson and Rishbeth (1988) used similarly sized inoculum segments singly to attempt inoculation of 32-year-old oak trees. Leach (1937) used a massive amount of inoculum to establish infection: large pieces of infected root were placed in a layer through which the roots of tea seedlings were allowed to grow. More recently Proffer and others (1988) used extremely large inocula relative to the size of the host: three stem segments 1.2 cm diam x 12-13 cm long were attached to the collar (approx 1 cm diam) of each 1-year-old experimental plant. As mentioned previously, the experiment gave unusually high levels of disease, an outcome which may have been partly due to the high inoculum potential resulting from the inoculation method.

As well as helping to increase inoculum potential, placing inocula close to the host may help to prevent disease escape. Rishbeth (1984), although working in an area and with species in which infection by rhizomorphs is probably the norm, considered it important to place inocula in contact with the host to allow the opportunity for non-rhizomorphic infection by isolates which are poor rhizomorph producers. In studies of species such as *A. tabescens* and *A. fuscipes* that normally infect only through root contacts, inocula are necessarily placed in contact with the host (Dadant 1963a, Plakidas 1941, Rhoads 1938, Rishbeth 1985b, Weaver 1974).

In some experiments with *A. tabescens* and *A. fuscipes* artificial wounds have been made at the point of inoculation (Dadant 1963a, Plakidas 1941, Weaver 1974). Wound inoculation has not commonly been employed with other species though Whitney and others (1989b) reported that such inoculations with *A. ostoyae* on fir roots were more successful than non-wound inoculations.

Rhizomorphs and Measurement of Disease in Inoculation Trials

Assessing virulence in trials has commonly been based on one or more of the following: amount of root infection, amount of mortality, or rapidity of infection or mortality. Such relatively straightforward measurements are, however, often complicated by the need to consider the role of rhizomorphs as extensions of the experimental inoculum.

Serious *Armillaria* diseases occur in several regions of the world where rhizomorph growth is restricted or absent (Dadant 1963a, Dade 1927, Kile 1981, Morrison 1981, Podger and others 1978, Rhoads 1956, Rishbeth 1980). Although non-rhizomorphic infection occurred commonly in Kile's (1981) inoculation trials with *A. luteobubalina* and Dadant's (1963a) with *A. fuscipes*, it has proved difficult to achieve experimentally with *A. tabescens*, the other economically important species known to infect in this way naturally (Rhoads 1956, Weaver 1974). Non-rhizomorphic infections by temperate species have occasionally been observed in inoculation trials (Rishbeth unpubl., Shaw 1977, Whitney and others 1989b) but some attempts to induce them deliberately have failed (Redfern 1978).

Most *Armillaria* experimentation has involved species in which rhizomorphs have been assumed to be the normal, or only, means of infection; most inoculation experiments have included a final assessment of the presence or absence of rhizomorphs. Among these studies are several reports of isolates which do not produce rhizomorphs readily, or at all, under experimental conditions (Gregory 1985; Mallett and Hiratsuka 1988; Rishbeth 1984; Rykowski 1981c, 1984; van Vloten 1936). Such isolates may eventually produce rhizomorphs given time (Patton and Riker 1959, Gregory unpubl.) or may be induced to do so by altering the method of inoculum production (Redfern 1970, Rishbeth 1968) or inoculum size (Benjamin and Newhook 1984b). Rhizomorph production, and hence disease, may also be strongly influenced by soil conditions (see chapter 4). Consequently, it may be difficult to decide whether lack of rhizomorphs, which is usually associated with lack of infection, reflects genuine field behavior or defective technique.

Interpreting results can be difficult in experiments where inocula in some replications produce rhizomorphs while those in others do not. Gregory (1985) treated such replicates as missing values, and Morrison's (1982b) scoring system also excluded replicates in which no rhizomorph contacted the host. However, some authors have included these data among non-infected categories, accepting the risk that this might distort results of trials with species that are poor rhizomorph producers.

Some of the problems associated with rhizomorph behavior are represented in the data of Mallett and Hiratsuka (1988), who found low disease levels and no rhizomorphs in trials with Canadian isolates of *A. ostoyae*. Since other evidence (discussed below) suggests that this species is a serious pathogen in both North America and Europe, the few infections achieved probably resulted not from low intrinsic pathogenicity but rather from the species' inability to produce rhizomorphs under the experimental conditions. European isolates of the same species have been characterized by Guillaumin and others (1985) and Gregory (1985) as poor producers of rhizomorphs in experiments.

Rhizomorph production may have a bearing on the optimum duration of *Armillaria* inoculation trials, a subject which has been briefly discussed by some authors (Benjamin and Newhook 1984b, Gregory 1985, Mallett and Hiratsuka 1988, Patton and Riker 1959), but which merits further attention. Several studies indicate that certain isolates take longer than others to cause visible, above-ground signs of infection (Gregory 1985, Raabe 1967, Redfern 1975, Rishbeth 1984, Wilbur and others 1972). The three isolates used by Wilbur and others (1972) differed little in virulence assessed simply as the proportion of experimental plants killed at the end of a 3-year trial. They would have been judged to differ markedly from each other, to the extent of one being almost non-virulent, had the final assessment been made after 18 months. Yet, this timespan equals or exceeds that chosen by many workers. In an unpublished trial using methods similar to those of Redfern (1975), Gregory found that 2 years after inoculation *A. mellea* had killed twice as many young conifers as *A. ostoyae;* however, after 3 years the position was reversed and was maintained until the trial ended 5 years after inoculation.

If a relatively slow rate of disease development reflects a relatively poorer ability of rhizomorphs contacting a host to initiate infection, then it may be a legitimate expression of lower virulence as some authors have proposed (Raabe 1967, Rishbeth 1984). If, by contrast, experimental manipulation adversely affects rhizomorph production and subsequently causes slow disease development, then the use of rate in comparative assessments is questionable. Guillaumin and others (1985) have noted that European species differ in the time taken to produce rhizomorphs under experimental conditions. They cite *A. ostoyae* as being especially tardy, an observation that coincides with unpublished data of Gregory and Rishbeth.

Most investigators who have studied pathogenicity in *Armillaria* have measured the amount of disease simply by the proportion of host plants killed or infected during the experiment. Several authors have used lesion size for scoring the severity of non-fatal infections (Gregory 1985, Guillaumin and Pierson 1978, Morrison 1982b, Rishbeth 1982). Assessments of dead or symptomatic plants have nearly always been visual and involved destructive examination. The main exception to the latter is a study by Zollfrank and Hock (1987), who conducted their experiments under aseptic conditions and used immunofluorescence to detect hyphae in seedling tissues.

Field Observation

The century-old descriptions of *Armillaria* disease by Robert Hartig (1874, 1894) reveal the field experience of a remarkable observer and stand comparison with modern accounts. From this beginning, field observations have been a major source of information about *Armillaria* disease, but they have also fueled much controversy over the role of *Armillaria* as a pathogen. *Armillaria* diseases are probably almost as difficult to observe critically in the field as they are to investigate by experiment. Worthwhile field observations require a comprehensive knowledge of forest pathology and of *Armillaria* biology as well as meticulous site investigation. Regrettably, some studies assume that the situations from which basidiomes have been collected fully circumscribe the ecology and pathogenic behavior of the fungus.

With our present ability to identify separate species of *Armillaria*, field observation has contributed significant information about pathogenicity. Despite Rishbeth's extensive experimental work, an appreciable proportion of our knowledge of pathogenicity in the European species derives from field observations (Gregory 1989; Guillaumin and others 1985; Guillaumin and Berthelay 1981; Korhonen 1978; Rishbeth 1982, 1984, 1985b). Field observations, notably those of Morrison and others (1985a), also constitute a major source of published data on North American species. In New Zealand and Australian studies, inoculation trials have complemented extensive field observations (Kile 1980b, 1981; Kile and Watling 1983; Pearce and others 1986; Podger and others 1978; Shaw and others 1981).

Indirect Methods of Assessing Virulence

Attempts to assess virulence indirectly have had only limited success. The idea of a direct relationship between virulence and host may be traced back to the observations Childs and Zeller (1929) made on what appeared to be a virulent "oak strain" of the pathogen and a non-virulent "fir strain." They were careful to acknowledge the danger of extrapolating their observations to other regions, but the idea of a link between host and virulence has persisted. However, despite having been investigated experimentally a number of times, no such connection has been demonstrated (Guillaumin and Pierson 1978, Raabe 1967, van Vloten 1936).

Possible relationships between virulence and the capacity to produce rhizomorphs have also received considerable attention. The apparent reliance on rhizomorphs for infection was taken by van Vloten (1936) to indicate that isolates which appeared to lack them were de facto non-virulent. Rykowski (1981c, 1984) observed good agreement between infection and rhizomorph production in his numerous experiments and used the relative abundance of rhizomorph growing tips as an index of "infection threat" in his three isolates, all of which

belonged to *A. ostoyae*. Some other studies involving single isolates or several isolates of the same species suggested a positive relationship between infection and rhizomorph production (Azevedo 1970-71, Guillaumin and others 1989a, Shaw 1977), but studies involving several isolates of widely different virulence have generally failed to demonstrate such a relationship (Guillaumin and Pierson 1978, Raabe 1967, Rishbeth 1984). Conversely, some evidence indicates a negative correlation of rhizomorph production to pathogenicity among European species (Gregory 1985, Redfern 1975, Rishbeth 1985b).

Morrison (1972, 1982b) and Redfern (1975) suggested an association between dichotomous branching of rhizomorphs and high virulence. The same authors also noted that highly virulent isolates tended to possess fragile rhizomorphs. We now know that Morrison's (1982b) three branching types represented three different species (Morrison 1989) and that Redfern's (1975) four isolates were also from four species (Gregory 1985). Later studies (Guillaumin and others 1985, Morrison 1989, Rishbeth 1982) have confirmed that branching habit and fragility of rhizomorphs are species characteristics. Morrison's (1989) data, drawn from 15 species, showed that a dichotomous branching habit (fig. 4.1) more often than not accompanied high pathogenicity but the association was not invariable. Three of the eight dichotomously branching species which he tested were of low pathogenicity. It may be unrealistic to seek universal relationships between growth patterns and pathogenicity among species that have evolved to survive in such widely different forest and soil conditions as have the various *Armillaria* species.

A few attempts have been made to assay virulence in *Armillaria* by in vitro characters. The most notable were based on the work of Wargo (1981d) that indicated a link between gallic acid metabolism and virulence in certain North American isolates. Shaw (1984, 1985) tested this hypothesis extensively on a collection of 72 isolates drawn from three continents. He found that although the ability to tolerate gallic acid varied among isolates, differences could not be utilized consistently as markers for virulence. Rishbeth (1986) reached a similar conclusion.

Differences in Pathogenicity and Virulence

Although taxonomists have for decades postulated the existence of several morphological species of *Armillaria* (see chapter 1), the recognition by pathologists of distinct pathogens has been comparatively recent. Two notable exceptions were provided by *A. tabescens*, which was accepted as a pathogen in its own right in the southern United States in the 1940's, and *A. fuscipes*, which Dadant (1963a) demonstrated to be a root pathogen of woody plants in Madagascar. Otherwise, before the late 1970's forest pathologists generally referred attacks of *Armillaria* disease to a single but variable taxon with worldwide distribution, *"Armillaria mellea."*

Some older data on the pathogenicity of *"Armillaria mellea"* have been reinterpreted relative to current taxa, but much information from before 1970 is of limited value. Modern studies of pathogenicity and virulence have concentrated largely upon North American, European, and Australasian isolates. Outside these regions, pathogenic species of *Armillaria* undoubtedly exist (see chapter 9), but little is known about the variation among them.

European and North American Species

Although forming a rather artificial grouping, these species are considered together because at least three, including the major pathogens *A. mellea* and *A. ostoyae*, appear to be common to both continents.

Evidence from inoculation trials identifies *A. mellea* as probably the most pathogenic species in this group. In Europe, isolates of this species have not only consistently been ranked highest in comparative studies but have also been demonstrated to cause disease in genera normally regarded as highly resistant to *Armillaria* (Davidson and Rishbeth 1988; Gregory 1985; Guillaumin and Pierson 1978*; Morrison 1982b*; Redfern 1975*; Rishbeth 1982, 1984). Three Canadian trials have included European isolates of *A. mellea* alongside North American isolates of other species, and in all cases the former have proved the most virulent (Mallett and Hiratsuka 1988; Morrison 1989, and pers. comm.; Mugala and others 1989). However, the results of inoculation experiments done by Guillaumin and Lung (1985) suggest that *A. mellea* may be less pathogenic than *A. ostoyae* to some conifers, an outcome which the authors interpreted as evidence of host specialization.

Field observations in Europe indicate that *A. mellea* is the most pathogenic species on ornamental trees, orchard crops, and vines (Guillaumin and Berthelay 1981; Guillaumin and others 1985; Intini 1988; Rishbeth 1982, 1985a). Even though it often kills ornamental conifers, and some isolates are extremely virulent toward young conifers in experiments, it is not widely associated with disease in forest or plantation conifers. In the United

*Isolates in Redfern (1975) were identified by Gregory (1985); those in Guillaumin and Pierson (1978) were identified by Guillaumin and Berthelay; those in Shaw (1977) were identified by Shaw (1984) and those in Morrison (1982b) were identified by Morrison (1989).

States, Proffer and others (1987, 1988) found that *A. mellea* was associated with root disease of cherry in Michigan, but few other observations on North American isolates involve this species.

In inoculation trials, North American and European isolates of *A. ostoyae* have generally been moderately or highly virulent towards young conifers (Gregory 1985; Guillaumin and Lung 1985; Morrison 1982b, 1989; Redfern 1975; Rishbeth 1982, 1984, 1985b; Shaw 1977; Siepmann and Leibiger 1989). Under experimental conditions, the species appears to be only weakly pathogenic to European forest hardwoods (Lung-Escarmant and Taris 1989, Rishbeth 1984). Rishbeth's (1984) data suggest that *A. ostoyae* could be classed with *A. gallica* as virtually non-pathogenic to common ash and silver birch although the isolates of *A. ostoyae* used were highly virulent to Scots pine in the same trial. Proffer and others (1988) found Michigan isolates of *A. ostoyae* to be highly virulent to *Prunus* species, but interpreting their results requires caution because of the equally high disease levels achieved with *A. gallica* isolates in the same experiments. Possible reasons for this have been discussed earlier in this chapter.

Isolates of *A. ostoyae* showing low virulence towards conifers have been reported in Europe (Rishbeth 1984), and recently, Mallett and Hiratsuka (1988) demonstrated apparently uniform low virulence toward young lodgepole pines in a range of Canadian isolates. As suggested earlier, such results may reflect the poor ability of some isolates to produce rhizomorphs under experimental conditions rather than innate low virulence. Indeed, *A. ostoyae* may be consistently underrated in inoculation studies for this reason.

Field observations in North America (Bloomberg and Morrison 1989, Dumas 1988, Harrington and others 1989, Mallett and Hiratsuka 1988, Morrison and others 1985a), Fenno-Scandia (Korhonen 1978, Piri and others 1990), and Europe (Gregory 1989, Guillaumin and Berthelay 1981, Guillaumin and others 1985, Intini 1988, Rishbeth 1985a, Siepmann 1985) indicate that *A. ostoyae* is a major forest pathogen of conifers in those regions. Several of these accounts show the species can kill trees of all ages and can also cause butt rot in older crops. So consistently has *A. ostoyae* been associated with disease in conifers that it is commonly assumed to be the probable pathogen whenever serious *Armillaria* disease is encountered in North American or European coniferous forests (Filip 1989a, Hadfield and others 1986, Hansen and Goheen 1989, Rizzo and Harrington 1988a, Whitney 1988b).

Despite the low pathogenicity towards hardwoods indicated by inoculation experiments, field observations suggest that *A. ostoyae* is capable of attacking broadleaved trees and shrubs growing within diseased conifer stands (Guillaumin and others 1985, Morrison and others 1985a, Rishbeth 1985a). Harrington and others (1989) recorded it as a cause of death of birch and maple in the northeastern United States, and the observations made in Canada by Dumas (1988) suggest that it may have a rather wide host range among hardwoods there, at least as a secondary pathogen.

Armillaria gallica has been widely categorized as a weak pathogen by both field observations and inoculations in Europe and North America (Gregory 1985, 1989; Guillaumin and Berthelay 1981; Guillaumin and Pierson 1978; Guillaumin and others 1985; Morrison 1989; Morrison and others 1985a; Redfern 1975; Rishbeth 1982, 1984; Shaw 1977, 1984; Siepmann and Leibiger 1989). Some isolates have been designated as virtually non-virulent (Rishbeth 1982, Shaw 1984), yet in few trials has this species completely failed to cause disease. In some cases (Guillaumin and Pierson 1978, Proffer and others 1988), it has caused appreciable damage, albeit to highly susceptible species. As already discussed, the level of damage achieved in trials with young, and possibly stressed, experimental plants may be artificially high. However, since newly planted crop or ornamental trees are also often young and stressed, it might therefore be unwise to dismiss comparatively weak pathogens such as *A. gallica* as harmless. Moreover, the ability of *A. gallica* to act as a secondary agent of mortality in large trees and to cause root- and butt-rot in live trees (Gregory 1985, Rishbeth 1982) implies a far from negligible capacity to cause disease. These remarks apply to most of the other species categorized below as weak pathogens.

Armillaria cepistipes is regarded in Europe as an analogue of *A. gallica*: a weak pathogen virtually indistinguishable from *A. gallica* in behavior and appearance (Guillaumin and others 1985). Few inoculation trials have been reported for this species, but those of Redfern (1975) and Morrison (1989) both indicated low virulence in the isolates tested. Rishbeth has unpublished data from the trials reported in 1985b, table 3, that also demonstrate low virulence. The species is nevertheless associated with butt rot of conifers in Finland and Scotland (Gregory 1989, Korhonen 1978, Piri and others 1990).

Of the six North American biological species (NABS) not clearly identified with European species (*A. gemina, A. calvescens, A. sinapina*, NABS IX, NABS X, and NABS XI), *A. sinapina* (NABS V) has probably received most attention because it is relatively common in some important forest areas (Mallett and Hiratsuka 1988, Morrison and others 1985a, Shaw and Loopstra 1988). The inoculation experiments with young trees in containers carried out by Morrison (1989), Mugala and

others (1989), and Shaw and Loopstra (1988) suggest that the species is of low pathogenicity towards some North American conifers. However, in another trial, Mallett and Hiratsuka (1988) found more infection caused in potted lodgepole pine by Canadian isolates of *A. sinapina* than by *A. ostoyae*. Although, as noted above, the latter may have been seriously underestimated in this experiment, the data for *A. sinapina* are nonetheless anomalous, the more so as Mugala and others (1989), using similar methods, reported low virulence towards the same host by one of the same isolates. Field observations in Canada support the view that *A. sinapina* is a weak pathogen (Dumas 1988, Morrison and others 1985a).

NABS IX also appears to have limited pathogenicity towards young conifers according to field observations and inoculation trials in British Columbia and Alaska (Morrison and others 1985a, Morrison 1989, Shaw and Loopstra 1988). Shaw and Loopstra (1988) found that haploid isolates of this species and *A. sinapina* caused significantly more disease than the parent isolates.

The observations of Morrison and others (1985a) in British Columbia placed the other northwestern species, NABS XI, in the same category as *A. gallica*, *A. sinapina*, and NABS IX. All are weak pathogens characteristic of suppressed or overmature hardwoods. The results of Morrison's (1989) subsequent inoculation trial with young Douglas-fir in pots supported this view since all four species received the same very low rating.

Armillaria gemina (NABS II) and *A. calvescens* (NABS III) were also included in Morrison's (1989) trial. Both were accorded the same low rating as NABS IX, NABS XI, *A. sinapina*, and *A. gallica*. Despite this, field observations on *A. calvescens* by Proffer and others (1987) in Michigan and by Harrington and others (1989) in New Hampshire associate it with root rot and mortality of hardwoods. In the case of *A. gemina*, Bérubé and Dessureault (1989) have stated that it is "identical to *A. ostoyae* in terms of . . . pathogenicity," but this view is based on extremely limited field observations. Little is known about the pathogenicity of NABS X, although McDonald (1990) suggests, again from limited observations, that it may be moderately pathogenic.

The northern European species *A. borealis*, which has not been recorded in North America, is generally regarded as a rather weak pathogen (Guillaumin and others 1985, Korhonen 1978), though observations from Britain suggest that some genotypes might be virulent to young conifers (Gregory 1989). Korhonen (1978) identified *A. borealis* as an important cause of butt rot of Norway spruce in Finland, and it has been associated with similar damage in Germany and Britain (Gregory 1989, Siepmann 1985). Only two inoculation trials have been reported. Both utilized young potted conifers; and both suggested that *A. borealis* is a rather weak pathogen, intermediate between *A. mellea* and *A. ostoyae* on one hand and *A. gallica* and *A. cepistipes* on the other (Morrison 1989, Siepmann and Leibiger 1989).

Although *A. tabescens* has been cited as causing root disease in trees in several parts of the world, considerable doubt now exists that a single species is involved (see chapter 1). Most information is available from the southern United States, where it is known as a serious pathogen of ornamental trees and commercial crops (Rhoads 1956, Sinclair and others 1987). The fungus can attack a wide range of woody species in a variety of genera but, according to Rhoads (1956), exotics are much more susceptible than native trees and shrubs. Rhoads (1956) also reported that damage caused by *A. tabescens* was particularly prevalent on drought-prone sites, and Weaver (1974) suggested that disease in peach only followed infection of previously killed or injured roots. Other reports associate *A. tabescens* with disease in stressed trees or trees primarily attacked by other agents (Filer and McCracken 1969, Ross and Marx 1972, Sinclair and others 1987).

A fungus referred to as *A. tabescens* has also been recorded in southern Europe as a root pathogen on several hosts including citrus on Corsica (Laville and Vogel 1984), eucalypts in southwestern France (Lung-Escarmant and others 1985a), and cork oak in Portugal (Azevedo 1976). Further north, European isolates of *A. tabescens* appear to be non-virulent in the sense of causing root mortality, though field observations have linked the species with decay of live trees in Britain (Rishbeth 1984, 1985b). The apparently southern distribution of diseases attributed to *A. tabescens* in both Europe and the United States is noteworthy because observations in China (Chang and others 1982) also associate severe root infection by *A. tabescens* with high soil temperature.

Australasian Species

Three Australasian species are regarded as serious pathogens on the evidence of field observation and inoculation trials: *A. luteobubalina*, *A. novae-zelandiae*, and *A. limonea*. Isolates of all three were represented in Morrison's (1989) trial which tested a range of European, North American, and Australasian species against 2-year-old Douglas-fir seedlings in containers. His results suggested that the three Australasian pathogens may be ranked with *A. mellea* and *A. ostoyae*. However, the amounts of disease recorded in most Australasian trials have been low by comparison to European or North American results with *A. mellea* and *A. ostoyae*. The contrast is particularly noteworthy in

similar tests on radiata pine conducted by Shaw (1977) in the United States with *A. gallica* and *A. ostoyae* and by Shaw and others (1980, 1981) in New Zealand with *A. novae-zelandiae* and *A. limonea).*

Field observations in New Zealand by MacKenzie and Shaw (1977) and Shaw and Calderon (1977) attributed disease in radiata pine crops to two native *Armillaria* species, *A. novae-zelandiae* and *A. limonea*, with the former appearing to be the more serious pathogen. Inoculation trials with young radiata pine in containers (Shaw and others 1980, 1981) demonstrated that both species were moderately pathogenic although some isolates of each had low virulence. Benjamin and Newhook (1984b) undertook trials with the same two species and found them highly pathogenic toward radiata pine, but in tests with eucalypts, *A. limonea* seemed to be less pathogenic than *A. novae-zelandiae.*

Armillaria novae-zelandiae also occurs in Australia where Kile and Watling (1983) recorded it as a secondary pathogen of native trees and a frequent cause of decay in myrtle beech. More recently, it has been cited by Kile and Watling (1988) as causing localized losses in young crops of exotic conifers, in which it is linked with *A. fumosa* and *A. pallidula*. Little else is known about either of these species though an isolate of *A. fumosa* was included in Morrison's (1989) trial in which it proved virtually non-virulent.

The chief Australian pathogen is undoubtedly *A. luteobubalina*. Field observations (Kile 1981, Kile and others 1983, Pearce and others 1986, Podger and others 1978, Shearer and Tippett 1988) have repeatedly demonstrated that it is a major primary pathogen in native sclerophyll forests where it kills eucalypts and a wide range of understory trees and shrubs. Infection can occur on eucalypts of all ages, resulting in crown dieback or mortality of large overstory trees as well as serious losses among seedlings and saplings. The fungus also attacks a wide range of species in vineyards, orchards, and ornamental plantings (Kile and Watling 1988).

Armillaria hinnulea by contrast was found to be weakly pathogenic in inoculation experiments with both native species and North American conifers (Kile 1980b, Morrison 1989). Morrison's (1989) data indicate that this species is similar to the European *A. borealis* in its ability to infect young Douglas-fir in containers. Field observations have characterized *A. hinnulea* as a weak pathogen capable of causing localized root lesions and decay in resistant hosts. It is nevertheless an effective secondary pathogen, and in this capacity, it is of some economic importance in Tasmania through association with "regrowth dieback," a decline of eucalypts of which the primary cause is unknown (Kile 1980b, Kile and Watling 1983).

Non-Australasian Tropical and Subtropical Species

Dadant (1963a) demonstrated experimentally that the morphological species he knew as *A. elegans* was pathogenic to field-grown albizia sp. His detailed observations and numerous isolations leave little doubt that the fungus he studied is a serious pathogen of coffee bushes and shade trees in Madagascar. Blaha (1978) associated the same fungus with damage to a similar range of hosts in Cameroon. The fungus is now known to occur widely in Africa and to be conspecific with *A. fuscipes* (see chapter 1), which was described by Petch (1923) as a root pathogen of acacia and probably also of tea bushes in Sri Lanka.

Most of the numerous accounts of *Armillaria* diseases in tropical and subtropical crops (see chapter 9) cannot now be validly attributed to morphological or biological species. However, the recent work by Mohammed and others (1989) with African isolates suggests that other pathogenic species in addition to *A. fuscipes* occur on that continent. Ironically, one of these appears to be at least partially interfertile with *A. mellea*—the name associated by default with disease in Africa since the early years of this century.

Conclusions

The genus *Armillaria* contains several virulent pathogens and other species that have evolved as successful secondary or facultative pathogens. Failure to appreciate this variation within the genus probably accounts for much of the controversy that has arisen in the past over the pathogenic status of *Armillaria*. Without doubt some species are primary pathogens, though the amount of disease caused by even the most pathogenic taxa may be conditional upon the nature of the host and the environment of the fungus. Most species appear to have a wide host range, but some species are apparently adapted to particular groups of hosts or site conditions or both. There is strong evidence that virulence differs among isolates of some species.

Experimentation with *Armillaria* poses formidable problems, and the interpretation of data from experiments and field observations is rarely straightforward. Nevertheless, our understanding has advanced remarkably rapidly in the past 20 years, though many aspects of pathogenicity merit further investigation. Despite the advances, relatively little is known about several North American biological species and even less about tropical and subtropical species.

CHAPTER 7

Host Stress and Susceptibility

Philip M. Wargo and Thomas C. Harrington

Armillaria root disease has historically been considered a disease of weakened trees. Early observers indicated that *Armillaria* was secondary to some other factor that predisposed trees to attack (Day 1927a, 1928, 1929; Falck 1918, 1923; Müller 1921; Nechleba 1915, 1927; Thomas 1934). Although not always the case, predisposition is considered common with Armillaria root disease, and seems to be more important in this disease than in the other woody root diseases of forest, shade, and orchard trees.

As with all diseases, susceptibility to Armillaria root disease depends on interactions among host, pathogen, and the environment. The importance of predisposing stresses and their impact on host vigor (the environmental component) must be considered in the context of the host and the pathogen. *Armillaria* has an extremely broad host range (Raabe 1962a), but these hosts vary in their susceptibility. Furthermore, many species of *Armillaria* are now recognized and these vary greatly in their pathogenicity (see chapter 6). Some are primary pathogens capable of killing vigorous hosts while others colonize only severely stressed individuals.

Stresses generally predispose trees to Armillaria root disease by reducing host vigor and, thus, compromising host defenses. Host defense mechanisms are addressed in chapters 4 and 5, but a brief review will set the stage for our discussion of stress and predisposition. Chronic and acute stresses and how they might affect resistance are covered in general, and specific examples of abiotic and biotic stress agents known to predispose trees to *Armillaria* are given. Lastly, we discuss forest management of Armillaria root disease relative to stress-induced susceptibility.

Stress Concepts and Host-Pathogen Interaction

Variation Among *Armillaria* Species, Host, and Site

Confusion about *Armillaria* taxonomy has hampered our understanding of stress effects on disease development. Unfortunately, very little research on stress-induced susceptibility has been conducted with known species of *Armillaria*. Where species of *Armillaria* have been identified, evidence suggests that root disease caused by *A. mellea*, *A. ostoyae*, or *A. gallica* is more likely to occur in a stressed host (Davidson and Rishbeth 1988).

Obviously, variation in pathogenicity among the *Armillaria* species (see chapter 6) has an important bearing on the requirement for a predisposing stress in disease development. *Armillaria gallica* only attacks stressed trees (Davidson and Rishbeth 1988) whereas *A. mellea* and *A. ostoyae* can infect and kill apparently vigorous trees. Stress may also broaden the host range of some *Armillaria* species. For example, *A. ostoyae* attacks primarily conifers but will also attack oaks when they are stressed (Davidson and Rishbeth 1988).

Predisposing stresses may be more important for disease development in relatively resistant species than in the more susceptible species. In general, hardwoods are considered more resistant to Armillaria root disease than coniferous species in northern temperate forests (Redfern 1978, Rishbeth 1972a). As discussed later, predisposing factors have been more often noted in Armillaria root disease on hardwoods than on conifers. However, *Armillaria* may be equally aggressive on healthy hardwoods, and this observation may reflect

the limited distribution of *A. mellea,* the species most capable of colonizing apparently healthy hardwoods (Davidson and Rishbeth 1988, Rishbeth 1982). Also, research on root and butt rots in hardwoods has been limited, and the disease may be more prevalent on hardwoods than commonly realized (Nordin 1954, Shigo and Tippett 1981).

Very limited information is available on resistance among hardwood species, but work on rootstocks of horticultural species shows that resistance varies both among and within species. Thomas and others (1948) reported that pear and walnut were quite resistant to *Armillaria*, but apricot and prune were susceptible. Variation in root stock resistance among several *Prunus* species was also reported in France (Guillaumin and Pierson 1983). Both studies demonstrated that peach and apricot root stocks were more susceptible to *Armillaria* than plum root stocks. Recent work by Guillaumin and others (1989b) verified that this relationship exists for *A. mellea sensu stricto.* The resistance of plum species was a dominant trait, and resistance to infection and colonization was maintained in some plum x peach hybrids.

Armillaria root disease occurs on many coniferous species (Raabe 1962a), but resistance varies considerably among and within species. In an English forest where Scots pine and Norway spruce were growing together, large patches of pine were killed while spruce were unaffected (Rishbeth 1972a). Inoculation studies on small trees, comparing resistance between conifers and hardwoods, showed that large differences existed among tree species in the percent of trees infected by *Armillaria,* and in the ratio of killed trees to surviving-infected trees; the hardwood species were generally the most resistant (Redfern 1978). Morquer and Touvet (1972b) also noted variation in resistance among conifer species, but no species tested was immune to infection.

Differences in resistance clearly occur within and among host species, but much of this observed difference may be related more to tree vigor than to genetic resistance. The importance of tree vigor in Armillaria root disease and the interplay of vigor and resistance make ranking of species susceptibility difficult, even with inoculation data (see chapter 6). Likewise, unless clonal material is available, identifying the importance of stresses and tree vigor is difficult.

Site factors and host adaptation play an important role in host vigor and susceptibility to Armillaria root disease. McDonald and others (1987a) found that the incidence of pathogenic *Armillaria* was low in habitat series of high productivity, unless the site was disturbed. In habitat series of low productivity, *Armillaria* was pathogenic in both disturbed and pristine sites. Disturbance was associated with increased disease incidence, but the association was weaker in highly productive sites where adaptive tolerances of the tree species were not exceeded. They suggested that Armillaria root disease was a problem on conifers in sites affected by human activities (including fire suppression), insects, or diseases, and in pristine sites where tree species were not adapted physiologically to their environment.

While little experimental work has been done to test this hypothesis, observations on where *Armillaria* is or has been a problem in forest stands tend to support it. For example, in the Northwestern United States *Armillaria* problems often occur in off-site plantations (Hadfield and others 1986, U.S. Dept. Agric. 1983) or transition forests that have been perpetuated by fire and disturbed recently by logging activity and fire control (Shaw and others 1976a). Problems with exotic species can also be related to maladaptation. Although these species may grow very well in new regions, they may not be well adapted to the climatic extremes in their new habitat. Consider, for example, radiata pine in high rainfall areas in New Zealand (Hawkins and Sweet 1989a,b). The factors important to site adaptation and tolerance of climatic extremes, including such physiological processes and conditions as net photosynthesis, cold and drought tolerance, and genetic variability, are also related to resistance to Armillaria root disease.

Host Vigor and Predisposition

The term "vigor" has been used to describe the overall robustness of a tree as indicated by its relative growth and absence of signs and symptoms of disease. Vigor is determined by a tree's physiological performance within a particular environment, and this performance depends upon the tree's genetic capacity. Genetic variation gives a range of physiological performances and therefore a range of physiological conditions or tree vigors under a given set of environmental conditions. Crown position (dominant, intermediate, or suppressed) and crown condition (good, fair, or poor) are commonly used to classify tree vigor. These are good indices of a tree's past relative growth and general vigor. However, they indicate little about a tree's current health and its vulnerability to the effects of stress (Wargo 1978a,b,c). When stressed by defoliation, for example, trees in all of the above vigor categories may be attacked and killed by *Armillaria* (Wargo 1977), indi-

cating that within these general vigor categories there are gradations of tree health. Herein, host vigor refers to the tree's current health and vulnerability.

Yarwood (1976) defines predispostion as "... the tendency of treatments and conditions acting before inoculation or before the introduction of the incitant, to affect susceptibility to biotic and abiotic pathogens." In the strict sense of this definition, trees are not always predisposed to infection by *Armillaria* since the pathogen may have already infected the roots prior to the stress. Many observations, especially in the *Armillaria*-hardwood relationship, suggest that for some combinations of hosts and *Armillaria* species the fungus rarely infects and colonizes an unstressed tree despite epiphytic pathogen growth on root surfaces (see chapter 8). Yarwood's broader definition of predisposition also includes changes that induce greater resistance to disease; however, only examples of increased susceptibility are emphasized in this chapter.

Predisposition to disease may play a much larger role in pathogenesis of forest-tree species than in other plant types because of their longevity. During the lifespan of a tree, it may be exposed to numerous stress-inducing episodes ranging from mild to acute and from short-term to chronic. Also, stresses that were inconsequential during a tree's early years can have devastating effects on the tree later. As trees increase in size and completely occupy their sites, their ability to maintain adequate moisture, nutrients, and energy levels approaches the physical limitations of the root and shoot systems; stresses can then cause considerably more damage.

Resistance to pathogenic organisms is the rule rather than the exception in forest trees. "If this were not so, they [trees] would have ceased to exist," (Shain 1968); or at least they would not live as long as they do. Although all trees have some capacity to resist infection, this resistance requires substantial energy. This metabolic energy is necessary to maintain or synthesize structural or chemical defenses that influence growth of pathogens at the surface of the plant or internally (Wood 1967). Production of physical and chemical barriers depletes the host's energy reserves, and trees of less than optimal vigor may not have the energy reserves required to resist infection and are therefore predisposed to disease. Conversely, host species with little genetic resistance will succumb if the pathogen is present, regardless of their energy reserves.

Stresses and Resistance to *Armillaria*

The term "stress" has been used to describe any environmental factor that can have potentially unfavorable influences on living organisms. Levitt (1972) defines "biological stress" as "any environmental factor capable of inducing a potentially injurious strain in living organisms" and "biological strain" as any change produced by the stress. The strain may be physical, such as the reduction of water flow through the transpiration stream in trees, or it may be chemical, such as a shift in carbohydrate metabolism.

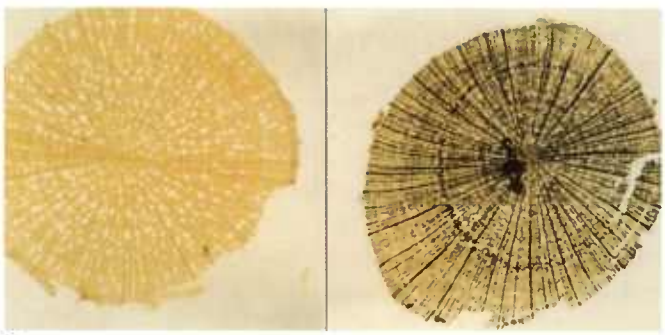
Chronic and acute stresses may disturb plants by altering resource allocation or by interfering with sink-source relationships (Waring and Patrick 1975). Stresses may interfere with the resistance response by reducing the energy reserves available for reaction (McLaughlin and Shriner 1980). Acute stresses may also temporarily impede metabolism at the infection site, and thus compromise the resistance response. The effects of a particular stress depend on severity, duration, season, frequency of occurrence, and the condition of the tree when it is stressed (Wargo 1978a,b).

Starch content has been used as an indicator of physiological performance and the effects of stress (Wargo 1978c). The susceptibility of stressed trees that are low or depleted in starch content probably relates, in part, to the reduced energy available for defense reactions (McLaughlin and Shriner 1980). For example, many oak trees are colonized by *Armillaria* after defoliation by the gypsy moth, but not all trees are infected, and not all infected trees are colonized to the same extent (Wargo 1977). Mortality of oak and sugar maple after defoliation was related to carbohydrate production and storage (Wargo 1981b,c,e; Wargo and Houston 1974). Trees with low or depleted starch when defoliation occurred were more likely to be colonized by *Armillaria* and to die after stress from defoliation (fig. 7.1). Starch content at the time of stress was related to how long a tree survived and how many defoliations it could tolerate.

Barriers and Energy Reserves

Preformed physical barriers such as outer bark play an important role in protecting roots from invasion by pathogens (see chapters 4 and 5). Outer bark may offer less protection from *Armillaria* than from those root-rotting fungi that cannot penetrate without wounds. Existing evidence does not suggest that predisposing stresses enhance susceptibility by allowing penetration through intact outer bark. However, some stress agents may cause bark injury and provide infection courts for *Armillaria*. Wind-induced root movements and breakage (Harrington 1986, Hintikka 1972, Rizzo and Harrington 1988b), rock abrasions (Stone 1977), and insect feeding provide infection courts for *Armillaria* and other root pathogens (Redmond 1957, Whitney

A



B

FIGURE 7.1 — *Armillaria* and energy reserves in roots of sugar maple. A: Sections of roots from defoliated (left) and non-defoliated (right) trees inoculated with *A. gallica* and incubated for four weeks; B: Starch reserves in roots from defoliated (left) and nondefoliated (right) trees in the fall after defoliation. Starch grains have been stained with I^2KI and appear purple-black in the tissue. (P. Wargo)

1961). Wounding and root breakage also stress trees since the tree must expend energy to close the wound, prevent infection, and replace damaged roots.

Wounds may not be so important in removing the barrier of the dead outer bark as they are in removing the living, responsive, inner bark. Once the outer bark is penetrated, the pathogen encounters living tissues where physiological factors, such as lytic enzymes or toxic secondary metabolites, may limit hyphal penetration of the inner bark.

The limitation of *Armillaria* hyphae developing within healthy host plant tissues has been described for the mycotrophic association between the fungus and achlorophyllous orchids (Hamada 1940, Kusano 1911, see chapter 8). In this relationship, lysis of the hyphae and reinfection by the fungus occur seasonally. The mechanism of hyphal lysis is unknown, but it could result from digestion by host enzymes. Chitinase and B-1,3-glucanase, enzymes that can dissolve the hyphal wall of *Armillaria*, are present in the inner bark and sap of forest tree species (Wargo 1975), and they constitute a potential mechanism to limit the growth of *Armillaria calvescens* hyphae in resistant bark tissue (Wargo 1975, 1976, and unpubl.). The activities of these enzymes are reduced by stress from defoliation (Wargo 1976).

An important component of the resistant reaction of the inner bark is the formation of wound periderms (Biggs and others 1984, Rykowski 1975, Thomas 1934). Some general observations indicate that stressed trees cannot produce periderms rapidly or fail to form wound periderms in response to *Armillaria* (Rykowski 1975). Even if they are formed, under some circumstances *Armillaria* has the ability to penetrate such suberized periderms (Rykowski 1975), probably by enzymatic degradation (Swift 1965, Zimmermann and Seemüller 1984).

Conversion of extant energy reserves into secondary compounds in response to wounding or invasion of inner bark or sapwood may benefit the host by forming compounds that are directly toxic to the pathogen, that are unavailable for pathogen metabolism, or that protect more complex carbohydrates from fungal extracellular enzymes (Worrall and Harrington 1988b). Gums, resins, phenolic compounds, and other metabolites may be produced in higher concentrations in response to wounding or invasion by pathogens than in unaltered sapwood (Hepting and Blaisdell 1936, Shain 1967).

Oleoresins in the inner bark and sapwood of conifers are potentially inhibitory to the fungus and are secreted in response to infection and colonization by *Armillaria*. Volatile components of oleoresin from Scots pine reduced the growth of *Armillaria* on agar by half (Rishbeth 1972a), and fewer rhizomorphs of *A. ostoyae* developed from resinous rootwood of Corsican pine than from non-resinous rootwood (Rishbeth 1985b). Roots of stressed conifers do not produce as much resin as healthy trees, and root tissues are colonized by fungi more rapidly than are roots of unstressed trees (Gibbs 1967, 1968; James and others 1980a,b; Rykowski 1975).

In spite of the emphasis on the role of the fungus as a phloem colonizer, *Armillaria* is capable of colonizing the inner wood of roots and stems without killing phloem tissues. This typical root- and butt-rot colonization may occur in relatively vigorous trees capable of resisting phloem colonization, and may proceed for decades without host mortality (Shigo and Tippett 1981, Tippett and Shigo 1981).

Two general sapwood responses are known (see chapter 5). First, sapwood tissues may be converted to non-living, reaction zone tissues that resist pathogen coloni-

zation (Shain 1967). Inhibitory, secondary compounds similar to those in the inner bark are also found in the reaction-zone tissues of the sapwood. As discussed in connection with the inner bark, these compounds require substantial energy reserves, and in stressed trees may not be produced in sufficient quantity or soon enough to stop *Armillaria* colonization.

Second, whether or not the pathogen becomes established in the reaction zone, another impediment to pathogen development, the barrier zone, may be formed. The cambium may respond by producing a unique layer of xylem that resists penetration by the pathogen and tends to restrict it to those growth rings of xylem formed prior to injury (Hepting and Blaisdell 1936, Shigo and Larson 1969). Barrier zones of this sort, formed in response to infection and colonization by *Armillaria*, have been observed in roots of both conifers and hardwoods (Shigo and Tippett 1981, Tippett and Shigo 1981).

Although evidence is limited, sapwood and cambium of less vigorous trees may form less inhibitory reaction zones and weaker barrier zones than the sapwood and cambium of healthy trees (Armstrong and others 1981, Shearer and Tippett 1988, Shigo and Hillis 1973). In such cases, *Armillaria* may be slowed but not stopped from developing in the sapwood, and continued development reduces the amount of sapwood available for water transport, increases the energy expended in resistance responses, and may allow penetration from the sapwood into the cambium and inner bark.

Pathogen Nutrition

Stress also affects resistance indirectly by nutritionally enhancing *Armillaria* growth. Predisposition of defoliated sugar maple to *Armillaria* occurs in part through changes in the carbohydrate and amino nitrogen compounds induced by defoliation (Wargo 1972).

Severe defoliation triggers hydrolysis of starch and results in large increases in reducing sugars in the cambial zone and neighboring tissues (Parker 1970, Parker and Houston 1971, Wargo 1972, Wargo and others 1972). Qualitative and quantitative changes in amino nitrogen also occur (Parker and Patton 1975, Wargo 1972) and, combined with increases in glucose, significantly stimulate the growth of *Armillaria calvescens* in vitro (Wargo 1972, 1981a, and unpubl.) (fig. 7.2). Hydrolysis of starch to glucose would certainly be more beneficial (nutritionally) to *Armillaria* than would conversion of starch to secondary metabolites, as would occur in the production of reaction zone tissues in healthy trees.

Stresses, such as excess soil moisture and defoliation, may also increase the ethanol in root tissues (Wargo unpubl.). Ethanol is a potent growth stimulant for *Armillaria* (Weinhold 1963) and its presence in root tissue could affect susceptibility to the fungus. The host may directly produce ethanol in response to stress (Coutts and Armstrong 1976, Crawford and Baines 1977); ethanol may be produced by associated microorganisms and promote the growth of *Armillaria* (Pentland 1967); or under anaerobic conditions, *Armillaria* may produce its own ethanol (Tarry 1969).

Chemical changes in roots of stressed trees apparently allow the fungus to metabolize phenols and probably other compounds that would normally inhibit it (Wargo 1980a, 1981d, 1983b, 1984a,b). Glucose, ethanol, and nitrogen levels and nitrogen source affect the ability of the fungus to oxidize phenols in vitro. Oxidation and polymerization of phenols by *Armillaria* can remove those that are inhibitory or that precipitate extracellular fungal enzymes. Also, phenol metabolism affects melanin formation by *Armillaria* (Bell and Wheeler 1986, Malama and others 1975, Worrall and others 1986) and could provide rhizomorphs and penetrating hyphae greater protection against enzymatic lysis from host-produced enzymes (Bloomfield and Alexander 1967). All of these host-pathogen biochemical interactions are discussed more fully in chapter 3.

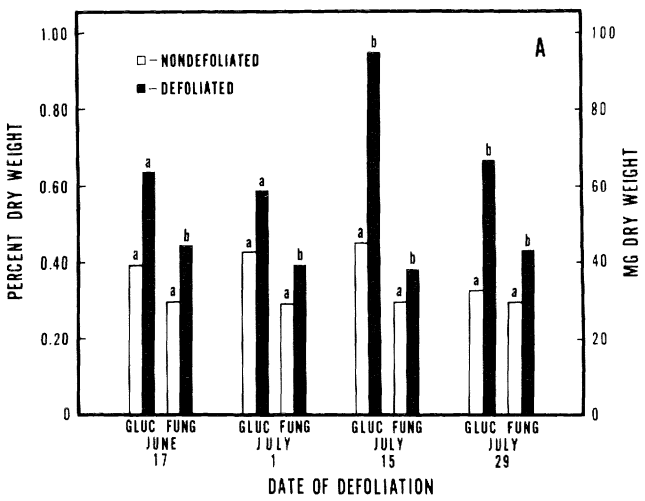


FIGURE 7.2 — Reducing sugar concentrations (% dry wt) in extracts from roots of defoliated and nondefoliated sugar maple, and fungal dry weight of *A. calvescens* after 3 weeks' growth on the extracts. Unlike letters above the bars indicate a significant difference at P=0.01. (Modified from Wargo 1972)

Stress Agents and Armillaria Root Disease

General

Trees are exposed to stress throughout their lives. Stresses such as drought, waterlogging, frost damage, some pollution events, insect defoliation, other tree diseases (especially foliar diseases), and short-term coppice cutting may be considered acute (short duration, high intensity). Other stresses may be considered chronic in that the tree may be exposed over its life time to low but relatively constant levels of the stress. Air pollutants, soil nutrient deficiencies, and long-term moisture deficiencies are examples of chronic stress. Shade-intolerant trees in forest understory can also be considered chronically stressed from reduced light.

Acute stresses may affect the metabolism of the entire tree, and *Armillaria* may rapidly colonize the entire root system or the root collar region of such trees and kill them quickly. Colonization of the roots of defoliated oak and sugar maple exemplifies this relationship (Wargo 1977, Wargo and Houston 1974). When acute stresses affect only a portion of the tree, *Armillaria* invasion may be partial and sometimes progressive, causing the tree to die slowly over several years. The relationship of *Armillaria* and beech bark disease demonstrates this interaction. *Armillaria* usually colonizes only those roots of American beech that are associated with the portion of the stem killed by *Nectria coccinea* var. *faginata* Lohman, Watson and Ayers, a canker-causing fungus (Wargo 1983a).

The timing of the stress event is also very important (Wargo 1978b). Stresses that occur early in the growing season and then abate have less of an effect than mid-season stresses because the trees have more of the growing season in which to recover. Likewise, stresses occurring late in the growing season may cause less harm because most of the growth and energy production by the tree has already occurred. The effects of any stress, no matter when it occurs, ultimately depend on its duration within and across growing seasons.

Stresses may also interact. Defoliation by phytophagous insects, especially those associated with oaks, have historically been linked to drought (Falck 1918, 1923; Houston 1981a,b, 1984; Nechleba 1915). These two stress factors working in concert affect tree health, resulting in widespread mortality, much of it associated with *Armillaria*. Defoliation can also exacerbate Armillaria root disease on beech affected by beech bark disease. On defoliated trees, *Armillaria* spreads from existing lesions on roots associated with the stem canker into adjacent roots and root collar tissues, resulting occasionally in rapid mortality (Houston 1974a, Wargo 1983a).

Abiotic Stress Factors

Light

Predisposition to Armillaria root disease from inadequate light has been observed in natural forests and plantations, and it has been demonstrated experimentally. *Armillaria* commonly attacks suppressed understory trees, upon which it acts as an ecosystem scavenger (Davidson and Rishbeth 1988, Pearce and others 1986, Rishbeth 1983). While these trees may be more susceptible to *Armillaria* attack because of genetic makeup, they are also affected by the reduced sunlight, which reduces the amount of energy available for defense against pathogens. Susceptibility, therefore, would be influenced by the shade tolerance of the tree species.

Redfern (1978) demonstrated the effects of insufficient light on susceptibility of trees to *Armillaria* in both plantation and greenhouse studies. Dominant and suppressed Scots pine in a 19-year-old plantation were inoculated and examined after 9 months. Similar numbers of dominant (12/15) and suppressed (13/15) trees were infected; however, the severity of infection, as measured by length of root invaded, was greater in the suppressed trees than in the dominant trees. Two suppressed trees were killed.

Inoculation studies with known *Armillaria* species on subdominant trees and suppressed trees growing in reduced light showed that suppressed English oak and Scots pine were infected by *A. mellea, A. ostoyae,* and *A. gallica* but not by *A. tabescens* (Davidson and Rishbeth 1988). Only *A. mellea* colonized the healthier subdominant oaks, and only *A. ostoyae* colonized the subdominant pine.

In one greenhouse study, *Armillaria* killed significantly more seedlings of Japanese larch growing under an 8-hr daylength than those growing under a 16-hr daylength for 20 weeks (Redfern 1978). In a second greenhouse study, seedlings of grand fir, western hemlock, and English oak were inoculated and grown under shade (70% light reduction) and compared with seedlings grown in full sunlight (Redfern 1978). Light did not affect susceptibility of western hemlock, which is a shade-tolerant species; 60% of seedlings were killed in each treatment. Shade, however, increased the susceptibility of the less-tolerant species, with 44% and 76% of the fir and 2% and 22% of the oak seedlings killed in full sunlight and shade treatments, respectively. Greenhouse studies with *A. ostoyae* on western

white pine also showed that very young seedlings (3-week-old) were more susceptible to infection if grown under reduced-light conditions (Entry and others 1986).

Temperature

Both high and low temperature extremes can stress trees and render them susceptible to opportunistic organisms. The effects of high temperatures, however, are commonly associated with drought, and distinguishing their individual effects can be difficult. In his report on environment and Armillaria root disease, Day (1929) indicated that the fungus attacked trees affected by sun scorch, drought, and defoliation. Sun scorch on leaves is caused by high temperatures associated with dry conditions and can significantly damage trees. Hole (1927a,b) found that drought and sun scorch on the foliage and sunscald on the smooth bark of morinda spruce in India significantly injured the crowns and predisposed the root systems to *Armillaria* colonization. Mortality was greatest on the hot, western and southern slopes and least on the cool, northern sites.

Elevated soil temperature, attributed to a slightly warmer summer climate and opening of the canopy by extensive logging, was proposed as a major factor in birch dieback in eastern Canada and Maine (Redmond 1955). Experimentally elevating the soil temperature by 2°C increased rootlet mortality from 6% to 60%. Trees in stands suffering "birch dieback" were characterized as having progressively greater rootlet mortality as crown vigor decreased. These trees were frequently colonized by *Armillaria*, but the fungus was not considered the primary cause of this decline (Hansbrough and others 1950, Spaulding and MacAloney 1931).

Bliss (1946) found that the greatest resistance to infection and colonization by *Armillaria* occurred at soil temperatures that were most favorable for root growth. Virulence was greatest at lower soil temperatures (10-18°C) on host species with a high soil temperature range for optimum root growth (17-31°C), such as peach, apricot, and geranium. On host species with a low soil temperature range for optimum growth (10-17°C), such as sweet orange, sour orange, orange and rose, virulence was greatest at higher soil temperatures (15-25°C).

Stress from freezing damage and subsequent colonization by *Armillaria* is documented for snowbrush. Severe crown kill of this evergreen shrub occurred in 1963 in Montana during a winter of light snow and after a sudden temperature drop from above freezing to -14°C to -20°C (Stickney 1965). A subsequent survey of snowbrush dieback in the Northwestern United States showed that *Armillaria* was associated with dead and dying clumps of this shrub (Tarry and Shaw 1966). Perhaps the freeze-damage predisposed the shrub to *Armillaria*. Subsequent work on this dieback (Tarry 1969) showed that 77% of the declining snowbrush stumps were infected by *Armillaria*. Results of inoculations in healthy plants were poor; less than 5% of 108 inoculation attempts resulted in infections, suggesting that colonization depended primarily on predisposing stress.

Infection and colonization of peach trees by *Armillaria* and other secondary organisms also were attributed (Poole 1933) to sudden exposures to low temperatures (-12°C to -9°C). These were extremes for peach orchards in the Carolinas (United States), and tree mortality ranged from 10% to 100%.

Damage from late spring frosts also predisposes trees to *Armillaria*. In North Carolina, late spring frosts were associated (Beal 1926) with the death of numerous white oaks. Later work indicated that much of this mortality was associated with *Armillaria* and bark insects (Hursh and Haasis 1931). *Armillaria* also infected chestnut trees (probably American chestnut) twice defoliated by late spring frosts (Long 1914).

Trees can also be stressed from events associated with but not directly related to low temperatures. Severe deterioration of an 80-year-old stand of red oak after a severe ice storm was attributed to *Armillaria* which colonized trees weakened by ice damage to their crowns (Dance and Lynn 1963). Hintikka (1974) suggested that Scots pine in plantations were predisposed to *Armillaria* infection by heavy snows that severely bent the saplings. However, *Armillaria* damage was severe in these snow-damaged plantations, perhaps due to increased wounding of the roots that lifted when the trees were bent rather than from direct reduction in tree vigor.

Moisture

Drought is probably the most common stress affecting trees, and at some time during most years trees experience either short- or long-term reductions in soil moisture. In their reviews on the relationship of *Armillaria* with widespread dying-off of forest stands in Europe, Twarowski and Twarowska (1959) and Nechleba (1915) indicate that attack of both conifer and hardwoods by *Armillaria* has been associated with drought since the late 1800's. Parasitism by *Armillaria* on true fir species was reported to increase during dry seasons, while wet seasons favored its saprophytic role (Nechleba 1927). Müller (1921) observed that droughts in the 1890's and early 1900's preceded *Armillaria*-caused deaths of many firs in Germany. Nechleba (1915) suggested that drought was the major factor in predisposing conifers to *Armillaria* and that the fungus "... under normal conditions of moisture and temperature, is a pronounced

and blameless saprophyte." Falck (1918, 1923) and Hen (1914) observed that drought was also involved in predisposing oaks to *Armillaria*. These early observations of stress-induced susceptibility to *Armillaria* led to the widely held view of the fungus as a secondary pathogen on forest trees.

Later reports also confirm the relationship of drought and *Armillaria*. Biraghi (1949) observed that infection of fir was enhanced during prolonged drought; however, mechanical injury also played a role. In East Africa, radiata pine were killed by *Armillaria* after an extended drought (Anon. 1952). In the United States, drought and subsequent *Armillaria* infection have been reported for western white pine (Ehrlich 1939), eastern hemlock (Secrest and others 1941), and balsam fir (Livingston and others 1982).

Oak decline and mortality in the United States have been frequently associated with drought conditions. Drought, in combination with defoliation from late spring frosts, followed by attack of the stressed trees by *Armillaria*, resulted in large-scale mortality in white, black, red, and scarlet oaks (Hursh and Haasis 1931). Staley (1965) also concluded that drought and defoliation from insects and frost damage predisposed scarlet oak to *Armillaria*. Similar relationships of drought, defoliation, and mortality of oak associated with Armillaria root disease were observed in Europe (Falck 1918, Hen 1914, Georgevitch 1926b). The European situation was further complicated by powdery mildew fungi that caused additional defoliation.

Drought also predisposes other hardwoods to *Armillaria*. The severe drought in the late 1950's through the mid 1960's in the Eastern United States was considered a predisposing factor in sugar maple decline. *Armillaria* afflicted 46% of symptomatic sugar maple trees in New York State in the early 1960's (Hibben 1964). Drought is also the most likely initiator of regrowth dieback of eucalypts in Tasmania where *A. hinnulea* and *A. novae-zelandiae* are important secondary pathogens (Kile 1980b, Kile and Watling 1983).

In a review paper on forest declines, Houston (1987) listed seven dieback and decline diseases, their episodic occurrence in North America since the early 1900's, and their associated stress factors and secondary organisms. Drought was listed as a stress factor in five of the seven diseases discussed; root-rot fungi, predominantly *Armillaria*, were involved in most of the declines. Other associations of drought, forest decline diseases, and *Armillaria* appear in table 8.3.

Root-system development may play some role in the predisposition effects of drought. Observations of Armillaria root disease on Scots pine indicated that root systems of healthy trees were deeper and better developed than those of infected trees. Susceptibility to drought and subsequent infection by *Armillaria* were favored in trees with a shallow, poorly developed root system (Ritter and Pontor 1969). Shallow roots and prolonged drought stress (7 years) were also associated with the decadence of eastern hemlock in Wisconsin (Secrest and others 1941). Declining trees were colonized by *Armillaria*, and root systems of some living trees with "normal" green crowns were also completely colonized by the fungus.

Excess moisture may be as stressful to trees as drought in regards to Armillaria root disease. However, the majority of such reports concern hardwood species. Excess soil moisture can cause physiological drought by interfering with water uptake in oxygen-deprived roots. Also, anaerobic conditions in the roots promote the production of ethanol, which can stimulate aggressive *Armillaria* growth (see chapter 3).

An early report on Armillaria root disease in the United States (Long 1914) indicated that *Armillaria* attack on various oak species and chestnut was greater and more severe on sites where the soil was wet seasonally. Wet summers also were observed to predispose chestnut species to *Armillaria* infection in Germany and Austria (Bazzigher 1956).

Native oaks in California were apparently infected with but not usually killed by *Armillaria* unless they were irrigated during the summer (Raabe 1966a). Whether irrigation resulted in excess soil moisture that stressed the trees or provided a better environment for more aggressive growth of *Armillaria* was not determined. Dade (1927) observed that high humidity promoted infection in cocoa. High rainfall years and poor soil drainage were also linked to infection of rubber trees in Nigeria (Fox 1964).

Decline of ohia has occurred periodically in Hawaii since 1875 and has been associated with poor soil drainage which, as the trees age, eventually predisposes them to *Armillaria* and other agents (Hodges and others 1986). In Japan, *Armillaria* on larch was related to low host vigor as indicated by annual growth increments, but incidence of infection depended mainly on the amount and duration of excess soil moisture (Kawada and others 1962). Disease was especially severe where larch were growing on soils with a high or perched water table.

Nutrients and Other Soil Factors

Armillaria root disease generally occurs more frequently and severely on nutrient-deficient soils or on

soils with poor physical and chemical characteristics for host growth. *Armillaria*-caused mortality in tea plantations growing in nutrient-deficient soils was considerably greater than in areas where soil fertility was adequate for growth (Butler 1928). In a 32-year-old plantation of eastern white pine in New York, damage by *Armillaria* was associated with low soil nutrients (Silverborg and Gilbertson 1961). Ono (1965, 1970) reported that *Armillaria* caused severe losses in Japanese larch plantations on both upper slopes and lowlands. In both areas, he attributed disease severity to physical and chemical soil characteristics unfavorable for larch.

Some evidence suggests that predisposition by nutrient deficiency depends on which tree species grows where a particular nutrient is low. Reduced nitrogen and phosphorus levels were linked to rapid development of Armillaria root disease in conifer plantations in Newfoundland (Singh 1970). Calcium deficiency was related to increased *Armillaria* damage in walnut plantations (Marchal and Foex 1931). Low soil nitrogen and low soil pH were associated with *Armillaria*-caused decay in Douglas-fir, while low soil calcium and phosphorus and high soil potassium were associated with *Armillaria*-caused decay in grand fir (Shields and Hobbs 1979).

Armillaria root disease has been related to extractable aluminum concentrations in soils from sites surveyed for root disease. Browning and Edmunds (1985) found that incidence of *A. ostoyae* on coastal Douglas-fir in the Northwestern United States was generally higher on sites where aluminum levels in the soil were low. Laboratory studies did not conclusively confirm this relationship (Browning 1987). Aluminum inhibited fungus growth but only at high concentrations in buffered media (200 ug/g and above). Fungal growth in coastal soil extracts decreased as extractable aluminum measured in these soils increased, but the correlation was not significant. Inoculated seedlings growing in soils from sites with high and low disease incidence also failed to associate disease incidence with extractable aluminum (Browning 1987).

Relationships between nutrients and susceptibility to *Armillaria* have been demonstrated experimentally. Rate, incidence, and severity of infection of seedlings of Norway spruce, black spruce, Sitka spruce, and Scots pine were greater when they were grown in forest soil with low nutrient levels and low pH (Singh 1983). Three-week-old seedlings of western white pine grown under reduced light and nutrient deficiencies were also infected more frequently and more severely than seedlings grown under adequate light and nutrient supply (Entry and others 1986). With adequate light, more seedlings that received nutrient solutions deficient in nitrogen or phosphorus were infected than those receiving the complete nutrient solution (Entry and others 1986).

Pollutants

Increased incidence and severity of Armillaria root disease associated with SO^2 and other pollutants have been observed (Grzywacz 1973, Jančařík 1961, Kudela and Novakova 1962, Novak and others 1957, Scheffer and Hedgcock 1955). However, reports associating Armillaria root disease with pollutants have been inconsistent, and generalizations are difficult. The influence of pollutants is related to the proximity of the forest to the source(s) of pollution. High pollutant levels nearer the source may inhibit the incidence of the disease, but more moderate levels may favor the disease.

In fluoride-damaged conifer stands in Newfoundland (Canada), the pollutant does not favor the disease. Singh and Sidhu (1989) found less Armillaria root disease in stands near an emission source than in stands farther away, and mycelial fans and rhizomorphs appeared less vigorous in the more polluted areas.

Grzywacz and Wazny (1973) observed that Armillaria root disease in Poland occurred two to three times more frequently in forests situated within or near industrial centers than in remote forests. Over an 8-year period from 1963-1970, area affected by Armillaria root disease expanded 3.5 times in forests near industrial centers compared to an overall forest increase of just 1.5 times. However, in young Scots pine plantations the percentage of trees attacked decreased as the proximity to the source and level of SO^2 increased (Grzywacz 1973, Grzywacz and Wazny 1973); incidence also decreased in forests beyond the zone of SO^2 influence. Thus, SO^2 pollution seemingly favors the disease except where the SO^2 levels are very high.

Later studies in Poland failed to support these results (Domanski 1978). He found that Armillaria root disease was extremely rare in polluted zones but was quite common in plots uninjured by pollution. Comparing the two studies (Grzywacz and Wazny 1973, Domanski 1978) is difficult because essential details are lacking in both. However, the differences may be related to the species studied, the age of the plantations, and the length of exposure to pollutants. Domanski (1978) suggested that *Armillaria* is suppressed in stands that have been exposed to air pollutants for long periods, but the disease is enhanced in stands that have been recently exposed to and weakened by pollutants.

Recently documented declines in forests of central Europe and eastern North America may or may not be

related to air pollution (Schütt and Cowling 1985, Worrall and Harrington 1988a), but *Armillaria* appears to be associated to some extent. Armillaria root disease occurs on some of the declining conifers in German forests (J. Worrall, pers. comm.). In a survey of mortality in spruce-fir forests of Crawford Notch and nearby Bartlett Forest, New Hampshire (United States), mortality attributed to *A. ostoyae* was frequently encountered at low, but not high elevations where pollution levels are higher (Harrington and others 1989, Rizzo and Harrington 1988a, Worrall and Harrington 1988a).

A survey for Armillaria root disease throughout the Northeastern United States found that *Armillaria* was associated with decline and mortality of red spruce, but incidence and severity of the disease decreased as severity of the decline and elevation increased (Carey and others 1984). These higher elevation sites are considered to be more polluted because of cloud precipitation (Lovett and others 1982, Scherbatskoy and Bliss 1984). The low incidence and severity of the fungus on declining and dead trees in the upper elevation forests was related to scarcity of rhizomorphs (Wargo and others 1987b). This paucity was correlated with high concentrations of lead (presumably from atmospheric deposition) in these upper elevation sites. Most of the isolates from these sites are *A. ostoyae* (Wargo 1989, and unpubl.).

Laboratory studies on *A. ostoyae* indicate that lead and other heavy metals present in soils of spruce-fir sites at high elevations inhibit both mycelial and rhizomorph growth in culture (Wargo and others 1987a). Rhizomorph production and growth were inhibited by both soluble and insoluble lead at concentrations found in soils at high elevations sites in the Northeastern United States. Inhibition was greater at lower pH levels, suggesting a potential interaction with soil acidification.

Disturbance from Partial Cutting

Partial cutting may intensify Armillaria root disease (Edgar and others 1976, Filip 1977, Filip and Goheen 1982, Kile 1981, Koenigs 1969, Redfern 1978). Release from competition should increase the vigor of residual trees, making them less susceptible. However, trees are often stressed upon initial release (so-called "thinning shock") and may succumb to Armillaria root disease before the benefits of release are established. Sunscald, winter injuries, wind stress, raised water tables, increased soil temperatures, and other environmental stresses may negatively affect residual trees, at least initially, and predispose them to Armillaria root disease. The problem of disturbance from cutting may be compounded because these weakened trees are surrounded by stumps which are food bases for the fungus.

Whether short-term stress from cutting predisposes the trees to existing inoculum or an increased inoculum potential causes increased disease is not clear. For example, western redcedar responded favorably, initially, to a thinning cut; however, 15 years later the residual trees were obviously in poor health (Koenigs 1969). Examination of the root systems of 45 trees indicated that 94% of the trees had rotted roots, and *Armillaria* was the most common fungus observed on or isolated from these diseased root systems. Conversely, residual red spruce in shelterwood cuts were colonized and killed by *Armillaria* within 3 years of cutting (B.Burns, pers. comm.), which would be too soon for appreciable mortality due to an increase in inoculum potential.

Partial cuttings in conjunction with other stresses can kill residual trees. On many sites in south-central Pennsylvania, shelterwood or seed-tree cuts in mixed oak stands followed shortly by gypsy moth defoliation resulted in complete loss of the residual trees (Wargo unpubl., and S. Cook, pers. comm.). These trees were attacked and killed by the two-lined chestnut borer (*Agrilus bilineatus* Weber) and *Armillaria* (Wargo unpubl.). Gottschalk (1989) showed that mortality in managed oak stands was equal to or higher than, but rarely lower than, mortality in unmanaged stands. Armillaria root disease and *Agrilus* attack were common on dead trees in these managed stands (Wargo unpubl.).

Partial cutting of red spruce in Northeastern United States also resulted in substantial mortality of the residual trees. These partial cuts were conducted during and shortly after the occurrence of severe droughts (1956-65). Pockets of *Armillaria*-induced mortality began to appear shortly after the cuts, and continued to expand through the early 1970's. These stands were overstocked, slow growing, and had no earlier thinning (W. Kingsley and B. Burns, pers. comm.). Subsequent cutting trials have indicated that where shelterwood cuts or heavy thinnings were conducted in overstocked, stagnated stands, severe mortality from *A. ostoyae* (isolates identified by Wargo unpubl.) struck the residual trees. If early thinnings were conducted, Armillaria root disease was not a problem on residual trees, either in subsequent commercial thinnings or in shelterwood cuts. Filip and others (1989) also reported that Armillaria root disease was not increased by precommercial thinning in ponderosa pine stands in central Oregon.

How partial cutting affects Armillaria root disease will likely depend on the site, the age of the stand when thinned or cut, the pathogenicity of the *Armillaria* species, and the health of the trees when cut.

Biotic Stress Agents

Insect Defoliation

The association of Armillaria root disease with defoliation is one of the best documented interactions. This relationship has been consistently observed and reported in forest studies. Also, defoliation has been documented experimentally to predispose trees; the mechanisms by which defoliation predisposes trees to *Armillaria* have been partially characterized.

Colonization of oak species by *Armillaria* after defoliation has occurred worldwide but especially in the United States and Europe. This may be related to both the number of oak defoliators and to several serious exotic insect defoliators that have caused widespread, severe defoliations. In Europe, the roles of *Armillaria*, defoliation, and drought were debated as the cause of widespread oak mortality by several workers (see review by Twarowski and Twarowska 1959). Mortality, primarily of English oak in England, Germany, and Yugoslavia, was related to Armillaria root disease and a number of oak defoliators, including insects and powdery mildew (Day 1927a; Falck 1918, 1923; Georgevitch 1926b; Yossifovitch 1926; Osmaston 1927; Robinson 1927). Most authors considered *Armillaria* to be a secondary pathogen.

In the United States, the association of *Armillaria* and defoliated oak has been noted since the early 1900's, and reports have increased in frequency since then. This increase has occurred because the importance of oak in the forest canopy has dramatically increased since chestnut blight, *(Cryphonectria (Endothia) parasitica* (Murr.) Barr) eliminated the American chestnut. Additionally, gypsy moths (*Lymantria dispar* L.) introduced into the northeastern United States in the late 1800's have caused widespread, severe, and repeated defoliations of oak. Attack of oak trees by *Armillaria* after gypsy moth defoliation was reported in Massachusetts by Baker (1941), but extensive tree losses after defoliation by the gypsy moth had occurred prior to this report (Burgess 1922) and most likely involved Armillaria root disease. Defoliation and hence mortality has increased as the gypsy moth infestation has expanded south and westward into areas of greater oak populations. Increased incidence of Armillaria root disease after defoliation has been reported in Connecticut (Dunbar and Stevens 1975), New Jersey (Kegg 1971, 1973), and Pennsylvania (Karasevicz and Merrill 1986; Karasevicz and others 1984; Nichols 1961, 1968). This process is occurring presently in Maryland, New York, West Virginia, and Virginia (Twery and others 1990, Wargo unpubl.).

Dunbar and Stephens (1975) suggested, based on presence or absence of the fungus at the root collar, that *Armillaria* played only a minor role in oak mortality after gypsy moth defoliation in Connecticut. Wargo (1977), however, showed that presence or absence of mycelial fans at the root collar did not indicate incidence and severity on the whole root system, and that *Armillaria* played a significant role in the mortality of defoliated oaks.

Defoliation by other insects also predisposes oaks to *Armillaria*. In Pennsylvania, Armillaria root disease was associated with decline and mortality of red and scarlet oaks defoliated by *Croesia (Argyrotoxa) semipurpurana* (Kearf.), the oak leaf roller (Staley 1965). In Bulgaria, *Armillaria* attacked oaks defoliated by leaf beetles (Shipchanov and others 1979).

Armillaria also plays a prominent role in the decline of defoliated sugar maples. A series of studies in Wisconsin on "maple blight" showed that defoliation initiated the problem (Giese and others 1964a,b). Ultimate mortality was often attributable to roots and root collars infected by *Armillaria* (Houston and Kuntz 1964). Armillaria root disease was also associated with sugar maple mortality in north-central New York after defoliation by the saddled prominent caterpillar, *Heterocampa guttavitta* Weber. (Wargo unpubl. and D. Houston, pers. comm.). Subsequent inoculation trials with an isolate of *A. gallica* on both artificially and naturally defoliated sugar maple showed that successful invasion of the root systems depended on stress from defoliation (Wargo and Houston 1974, Wargo unpubl.).

Armillaria attack after defoliation has also been reported for conifers. In Canada, defoliation by the spruce budworm, *Choristoneura fumiferana* (Clemens), apparently predisposes balsam fir (Sterner 1970, Stillwell and Kelly 1964) and black spruce (Raske and Sutton 1986) to *Armillaria*. Raske and Sutton (1986) found that infection increased from 30% to 85% when defoliation exceeded 80%. Filip (1989b) reported a very low incidence of Armillaria root disease in grand fir stands in Oregon that had been defoliated heavily for three years by the western spruce budworm, *Choristoneura occidentalis* Freeman. Based on inoculation studies, he suggested that the involved species of *Armillaria* was not very pathogenic. Increased Armillaria root disease also was associated with defoliation of western larch by the larch case bearer (*Coleophora laricella* Hubner) in Idaho (Tunnock and others 1969) and defoliation of Norway spruce by *Epinotia nanaxa* Treitschke in Norway (Austara 1984).

Another form of "defoliation" that occurs periodically is short-rotation, continuous cropping of trees such as that used in aspen management (see chapter 8). Defoliation is sudden and complete, and the tree responds

by producing stump sprouts that perpetuate the root system. The incidence of *Armillaria* increases with the number of successive coppices (Stanosz and Patton 1987a,b; Stiell and Berry 1986).

Other Insects

Increased incidence of *Armillaria* is associated with insects other than defoliators. In Newfoundland (Canada), disease incidence and severity markedly increased in balsam fir stands infested by *Adelges piceae* (Ratz.), the balsam wooly adelgid, a sap-sucking insect (Hudak and Singh 1970, Hudak and Wells 1974). The number of trees infected by *Armillaria* and the severity of infection were directly proportional to the severity of damage by the wooly adelgid.

Beech trees are predisposed to *Armillaria* when they are attacked by *Cryptococcus fagisuga* (Linder.), the beech scale. In this case, the scale predisposes the stem bark to a canker fungus, which then predisposes roots to infection by *Armillaria*.

Hylobius root weevils, *Hylobius warreni* Wood and *H. pinicola* Couper, also have been reported to predispose conifers in Newfoundland to Armillaria root disease (Warren and Singh 1970). Incidence of root disease increased in weevil-injured versus uninjured trees for Sitka spruce (15% vs. 4%) and Norway spruce (7% vs. 5%). In red pine, incidence was low and somewhat less (1% vs. 3%) in weevil-injured trees. Because feeding wounds made by these weevils may be important infection courts for *Armillaria* (Warren and Whitney 1951, Whitney 1961), the association with the weevil may not be predisposition in the same sense as with the aforementioned defoliators.

A reverse association of Armillaria root disease and bark beetles occurs among the conifers. In these relationships, root diseases, including *Armillaria*, stress the trees and predispose them to attack, colonization, and subsequent killing of the tree by bark beetles (Cobb 1989, Cobb and others 1974, Kisielowski 1978, Maslov and Nizharadze 1973, Secrest and others 1941, Thomas and Wright 1961). Such attacks may be important for maintaining endemic beetle populations (see chapters 8 and 10).

Trees with root disease may not just be more susceptible to successful beetle attack, but also may be more attractive to the insects. Increased production of volatile oils and changes in the chemical makeup of oils in needles of Norway spruce occurred in trees colonized by *Armillaria* and subsequently attacked by *Ips typographus* (L.) (Madziara-Borusiewicz and Strzelecka 1977). At least one volatile oil, myrenetol, is related to attractants and aggregation-pheromone production in bark beetles other than *I. typographus*.

The associations of bark-infesting beetles and *Armillaria* in hardwood trees have not been studied as intensively as in conifers. The two-lined chestnut borer *(A. bilineatus)*, which attacks most oaks and various other hosts, commonly attacks trees stressed by drought and defoliation, and is therefore commonly associated with Armillaria root disease (Cote and Allen 1980; Dunbar and Stephens 1975, 1976; Kegg 1971, 1973; Nichols 1968; Staley 1965; Wargo 1977). Various roles in tree mortality were assigned to each organism based on its incidence and severity of attack. Both organisms, however, contribute to mortality after stress; and both *Armillaria* and the borer can attack trees independent of each other or in combination (Wargo 1977). Unlike the conifer relationship, Armillaria root disease does not commonly predispose oaks to attack by the two-lined borer (Wargo 1977).

Other Diseases

Armillaria occurs commonly with other root pathogens in conifer stands, especially *Phellinus weirii* (Murr.) Gilb., *Heterobasidion annosum* (Fr.) Bref. *(Fomes annosus)* and *Phaeolus schweinitzii* (Fr.) Pat. (Filip 1979, Filip and Goheen 1984, Hansen and Goheen 1989, Hobbs and Partridge 1979, Whitney and Myren 1978; see chapter 8). In many infection centers, *Armillaria* occurred with one or more root pathogens (Goheen and Filip 1980). The pathogens colonized roots of adjacent trees and, in some cases, roots of the same tree. *Armillaria* was commonly associated with *P. weirii, H. annosum*, or *Leptographium (Ophiostoma) wageneri* (Kendr.) Wingf. on grand fir, Douglas-fir, lodgepole pine, and ponderosa pine (Filip and Goheen 1982, Goheen and Filip 1980).

These associations among root pathogens could be coincidental or the consequence of successional relationships. *Armillaria* may colonize Douglas-fir infected with *P. schweinitizii* in the U. S. Pacific Northwest (Hansen and Goheen 1989), but the reverse order of colonization was reported in Britain (Barrett 1970, Barrett and Greig 1985). *Leptographium wageneri*, the causal organism of black stain root disease in conifers in the Western United States, seemed to predispose ponderosa pine, Douglas-fir, and mountain hemlock to *Armillaria* (Goheen and Hansen 1978). Armillaria root disease occurred only occasionally at margins of disease centers of black stain root disease, but occurred frequently within the infection centers on trees affected by *L. wageneri*. Similarly, Byler and others (1983) found *Armillaria* on black-stained Douglas-fir within infection centers, but only the black stain fungus on trees at the margins of the centers.

Ceratocystis virescens (Davids.) C. Moreau, the causal organism of sapstreak disease of sugar maple (Hepting 1944, Houston and Schneider 1982, Kessler and Ander-

son 1960), appears to predispose sugar maple to *Armillaria*. Hepting (1944) found Armillaria and Xylaria root disease fungi commonly on trees with sapstreak disease. The sapstreak pathogen produces abundant volatiles on colonized sugar maple wood; these materials stimulate *Armillaria* growth in vitro (D.R. Houston, pers. comm.).

Foliage diseases can weaken trees by reducing or eliminating leaf surface area available for photosynthesis. Falck (1918, 1923) reported that English oaks in Europe were attacked and sometimes killed by *Armillaria* after they had been defoliated by *Microsphaera quercina* (Schw.) Bunill, the powdery mildew fungus. The trees had been stressed by earlier insect defoliation and drought, and had refoliated; new leaves are susceptible to mildew attack and complete defoliation by the fungus.

In New Zealand, growth loss of radiata pine was related to a combination of needle blight caused by *Dothistroma pini* Hulbary and root disease caused by *A. novae-zelandiae* or *A. limonea* (Shaw and Toes 1977). Sample size and method precluded clarifying the predisposition roles of each organism. However, growth loss of trees attacked by both organisms was greater than the growth losses attributable to each organism alone; only trees infected by *Armillaria* died. This suggests that severity of *Armillaria* attack was enhanced by needle blight.

Beech trees in northeastern North America are, as noted earlier (Ehrlich 1934, Wargo 1983a), predisposed to *Armillaria* attack by beech bark disease (fig. 7.3). Roots associated with stem portions killed by *Nectria coccinea* var. *faginata* are commonly attacked by *Armillaria*. If attack by the scale and canker fungus continues circumferentially, additional roots are attacked by *Armillaria*. This relationship can continue until eventually the tree is killed, girdled above by the canker fungus and below by *Armillaria*. However, progression of the canker disease may cease because of reduced scale populations. In these cases, *Armillaria* becomes established as a decay organism on the initially infected roots but is prevented from colonizing adjacent healthy tissues by vigorous callousing.

Blister rust, caused by *Cronartium ribicola* Fisch., can predispose western white pine to *Armillaria*. Kulhavy and others (1984) found high correlations between percentage of roots infected by *Armillaria* and bark beetle attack, and between percentage of crown killed by *C. ribicola* and bark beetle attack. The authors hypothesized that trees invaded by blister rust were predisposed to Armillaria root disease, which in turn predisposed trees to attack by bark beetles.

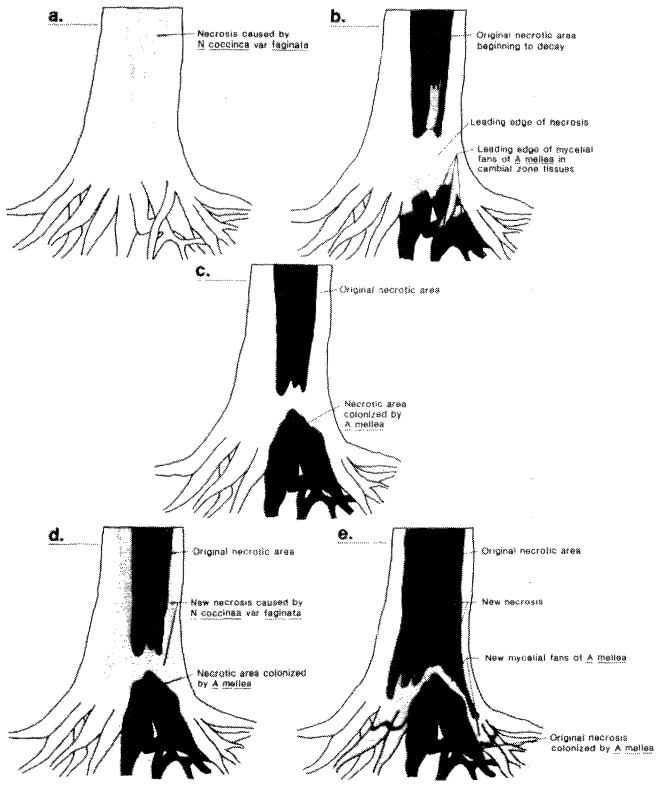


FIGURE 7.3 — Diagram of the lower stem and root-stem base of a beech tree, illustrating the timing and pattern of stem colonization and subsequent necrosis caused by *Nectria coccinea* var. *faginata* and corresponding colonization of the roots by *Armillaria*. a: Initial stem necrosis caused by *Nectria*; b: Necrotic area on roots caused by *Armillaria*; c: Necrotic area on stem in advanced stages of decay; necrotic area in roots completely colonized by *Armillaria* and beginning to decay; d: New stem necrosis caused by *Nectria* adjacent to original necrosis; e: Invasion by *Armillaria* of roots corresponding to area of new stem necrosis (from Wargo 1983a).

Another major tree disease that predisposes conifers to *Armillaria* is dwarf mistletoe (*Arceuthobium* spp.). *Armillaria* and *H. annosum* were associated with dwarf mistletoes in causing mortality on true firs and pines (Byler 1978). Root pathogens, primarily *Armillaria* and *H. annosum*, in combination with dwarf mistletoe, accounted for 11% to 28% of overall conifer mortality found in surveys in four California national forests (Byler 1978). In Manitoba, Canada, accelerated mortality of jack pine in localized centers in stands affected by dwarf mistletoe is attributed to Armillaria root disease (T. Meyer pers. comm.). The problem is concentrated on poor sites with deep sandy soils.

Managing Stress

Controlling dieback and decline diseases that are stress-initiated and involve *Armillaria* focuses on reducing or preventing the effects of the predisposing stress (Houston 1973, 1974b, 1981c). Alleviating or preventing predisposing abiotic stresses such as drought, frost, and waterlogging may be difficult in a forest setting. However, in urban, park, and garden settings, watering (but not overwatering), fertilizing, pruning, mulching, and proper site selection can reduce or eliminate the effects of temperature and moisture extremes. These practices can reduce the chances for infection and colonization by *Armillaria*. For some biotic stress agents, direct control to prevent insect infestations or disease buildup will eliminate the stress and reduce or prevent colonization by *Armillaria*. Direct control of defoliators, such as the spruce budworm or gypsy moth, by spraying insecticides should ultimately reduce mortality caused by *Armillaria*.

Silvicultural practices can be used to regulate species composition, maintain biological diversity, reduce chances for insect pest buildup on selected tree species, and increase host vigor (Houston 1981c). For example, silvicultural techniques could reduce the susceptibility and vulnerability of stands to beech bark disease by reducing the stand's beech component, while at the same time retaining beech trees that are resistant to the beech scale (Houston 1981c). Managing oak forests to control gypsy moths can also lead to reduced Armillaria root disease. Forests that are most resistant to defoliation are those with diverse species compositions growing on mesic sites (Houston and Valentine 1977, Valentine and Houston 1979). Maintaining diversity through forest management ensures the perpetuation of forests more resistant to defoliation, and these low-stress forests should be more resistant to Armillaria root disease. Partial cutting or thinning may also increase host vigor and resistance to Armillaria root disease (see chapter 11). However, as mentioned earlier, partial cutting may stress residual trees and lead to more Armillaria root disease in some forest types, so cutting practices may need to be altered.

Conclusions

Predisposing stresses significantly affect the development of Armillaria root disease. Even where *Armillaria* functions as a primary pathogen, stress may have some as yet undefined role in disease development. A wide variety of both abiotic and biotic factors may stress a host tree and allow infection and colonization by *Armillaria*. Limited evidence suggests that stress impairs physiological processes critical to resistance and decreases the energy reserves required to sustain the resistance response. At the same time, stress-induced chemical changes provide the fungus with abundant carbohydrate and nitrogen sources, and perhaps other nutrients, that stimulate vigorous growth of *Armillaria*. Alleviating the stress should control Armillaria root disease, perhaps by allowing the host to fully express its genetic capabilities to resist infection.

Our understanding of stress-induced susceptibility to *Armillaria* is limited by information regarding distribution of *Armillaria* species, understanding the physiological and pathogenic capabilities of each species, and recognizing the different relationships among various host and *Armillaria* species. We particularly need information about which combinations of pathogen and host have an essential requirement for predisposing stresses, which combinations require no stress to cause disease, and in which combinations disease is merely enhanced by stress. Inoculation studies using several genotypes of each *Armillaria* species and clonal host material, performed in both controlled and natural environments, may provide this information. Species of *Armillaria* must also be identified when disease episodes associated with various stresses are investigated. The concepts presented in this chapter undoubtedly will change as we increase our knowledge and understanding of *Armillaria* species, and of their relationships with host species and climates throughout the world.

CHAPTER 8

Ecology and Disease in Natural Forests

Glen A. Kile, Geral I. McDonald, and James W. Byler

Armillaria is unique among the basidiomycete genera that include woody root- and butt-rot parasites. It occurs worldwide in boreal, temperate, and tropical forests, and through diverse parasitic activities it affects a broad variety of host species. Species of the genus are, therefore, a significant consideration in the ecology and management of many natural forests.

Armillaria (as *A. mellea*) was first recognized as a pathogen in plantations and amenity plantings (Hartig 1873b, 1874). Initially, the fungus was often considered to be purely an opportunistic pathogen infecting plants weakened by other biotic or abiotic agents (Day 1929). While Day clearly realized the potential for both secondary and primary pathogenic behavior, he also stated, "It is quite possible that in natural forest the fungus frequently acts in this second (i.e., primary) role, but if that has been observed it does not appear to ever have been recorded." Only in the last 25 years have several *Armillaria* species received wider recognition as important primary pathogens in some natural forests.

Disease in natural forests significantly impacts forest economics, and forest harvesting and management activities may aggravate the endemic disease caused by *Armillaria* species. To minimize disease losses, forest managers must understand the ecology of *Armillaria*. This understanding also improves knowledge of disease development in plantation and amenity plantings on ex-forest sites. The incidence and severity of disease in the former is initially determined by the *Armillaria* species present and its distribution in the primary community. More broadly, the study of *Armillaria* in forests can enhance our general understanding of disease development in wild populations (Burdon 1987).

This chapter examines the ecology and parasitic behavior of *Armillaria* species in natural forests, disease impacts, and the influence of environmental factors and forest management activities on disease expression.

Geographical Distribution of Species

Armillaria is a natural component of the mycoflora of many forests worldwide. The genus has been most intensively studied in temperate regions, and observations and disease records suggest that more species occur and are more abundant in temperate and boreal forests than in tropical forests. Within the latter zone, *Armillaria* appears most abundant and frequent in forests above 500 m although species also occur in the lowlands (Fox 1964). Although the precise altitudinal and latitudinal limits for the genus have not been defined, *Armillaria* is restricted by excessively wet, cold, or dry conditions. These factors also limit host distribution, but not necessarily to the same extent. In western North America, hosts may grow on arid sites where *Armillaria* may be absent (McDonald and others 1987b). This may reflect either the physical environment which prevents infection or survival or, alternatively, that the distance between hosts does not allow spread even if the fungus were to become established.

The best documented geographical distributions are for five *Armillaria* species in Europe (fig. 8.1). For most species, however, distributions are incompletely known. As a consequence of more recent taxonomic studies and better understanding of the ecology of some species, such information can be expected to increase in the future.

Natural distributions are likely to reflect species origins, opportunities or fitness for long-distance dispersal, or adaptation to a particular host or forest type over a long period. Consistent associations are now recognized for a number of species. These include *A. mellea* and *A. ostoyae* present in various hardwood and coniferous forests, respectively, across the northern hemisphere, and *A. borealis* apparently restricted to high-latitude coniferous forests in Europe and Russia (Anderson and others 1980; Guillaumin and others 1985, 1989a; Rishbeth 1982; Terashita and Chuman 1987, 1989). In Australia, Kile and Watling (1983, 1988)

FIGURE 8.1 — Recognized distribution of five *Armillaria* species in Europe (updated from Guillaumin and others 1985).

identified four species associated with different forest types or ecological situations (fig. 8.2). One species, *A. novae-zelandiae,* found in temperate rainforests in eastern Australia, also occurs in New Zealand and possibly South America (Kile and Watling 1983, 1988; Singer 1969). This suggests a long link with southern-beech and other temperate rainforest species.

Within such broad distributions, factors such as altitude may further differentiate species occurrence. In Europe, *A. mellea* and *A. gallica* are regarded as low-elevation species, while *A. cepistipes* occurs at higher elevation (Guillaumin and others 1989a).

In other forested areas, clear patterns of species distribution have not yet emerged and a number of species may coexist. Little data is available for Africa, South America, parts of North America, China, or Siberia.

Although the extension of *Armillaria* species distributions through trade or introductions of infected planting material is possible (Pegler 1986, Piper and Fletcher 1903), no documented example of species naturalization resulting from human activities exists.

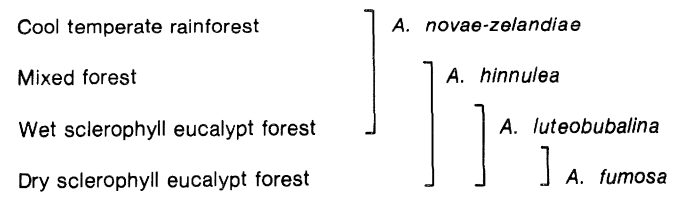


FIGURE 8.2 — Occurrence of *Armillaria* species in major forest types in southeastern Australia, established from basidiome collections during the period 1974-1981. (From Kile and Watling 1983, reproduced courtesy of British Mycological Society).

Host Range

Collectively, species of *Armillaria* have a very broad host range within the native vegetation where they occur. A large host list has been published for *A. mellea* (Raabe 1962a, 1979a), but attribution of hosts on a worldwide basis to this single species now requires revision. Given our expanding knowledge of *Armillaria* species, we are only beginning to determine the host range of many species in their natural communities, a salutory consideration more than a century after Hartig (1873b) identified *A. mellea* as a parasite.

The nature of the task may be illustrated by *A. luteobubalina,* an Australian species first described in 1978 (Podger and others 1978) and currently one of the few species for which it is possible to prepare a reasonably comprehensive host list. In those forests where it occurs, *A. luteobubalina* infects 81 species in 21 families, including monocots and dicots, and species in each forest strata (table 8.1). The list expands when hosts introduced to Australia are considered (Kile and Watling 1988, Smith and Kile 1981). This emphasizes the continued need for recording hosts so we can fully understand behavior of *Armillaria* species.

Factors determining host preference or specialization in natural forests or whether such phenomena can be clearly defined requires further assessment. Current understanding partly reflects past confusion over species identity, but the issue is undoubtedly complex. Disease caused by the same *Armillaria* species may be expressed differently on various hosts in the same community (e.g., root rot, butt rot, killing). Some species considered pathogenic on hardwood or conifers, respectively, may opportunistically infect both tree types while others can routinely infect plants in both groups. Stress may extend the host range of some species. Details of host specialization/preference are discussed in chapters 4 and 6.

Modes of Behavior in Natural Forests

As facultative necrotrophs, *Armillaria* spp. kill living tissues, then utilize them as a nutrient source. Some species may be obligate saprotrophs, but all species investigated to date appear to have some capacity to infect at least stress-weakened but living host tissue. The generally moist forest environments in which they are active, and defense mechanisms such as pseudosclerotial plates and antibiotic production by which species retain control of infected material, may extend

TABLE 8.1 — Hosts of *Armillaria luteobubalina* in various strata of dry sclerophyll eucalypt forest in Australia.

Family	Host Species
	Overstory
Myrtaceae	*Eucalyptus baxteri* (Benth.) Maid. & Blakely
	E. calophylla Lindley
	E. camaldulensis Dehnh.
	E. cypellocarpa L. Johnson
	E. diversicolor F. Muell.
	E. dives Schau.
	E. globulus (Labill.)spp. bicostata (Maid. et al.) Kirkp.
	E. gomphocephala DC.
	E. gummifera (Gaertn.) Hochr.
	E. macrorrhyncha F. Muell. ex Benth.
	E. marginata Donn: ex Smith
	E. melliodora A. Cunn. ex Schau.
	E. obliqua L'Herit.
	E. ovata Labill.
	E. patens Benth.
	E. radiata Sieb. ex DC.
	E. rubida Deane & Maid.
	E. rudis Endl.
	E. viminalis Labill.
	E. wandoo Blakely
	Understory
Casuarinaceae	*Allocasuarina fraseriana* (Miq.) L. Johnson
	A. huegeliana (Miq.) L.Johnson
	A. humilis (Otto & Dietr.) L. Johnson
	Casuarina decussata Benth.
Compositae	*Cassinia aculeata* (Labill.) R. Br.
	Olearia argophylla (Labill.) Benth.
Mimosaceae	*Acacia dealbata* Link.
	A. extensa Lindley
	A. mearnsii De Wild.
	A. melanoxylon R. Br.
	A. pulchella R. Br.
	A. saligna (Labill.) H. L. Wendl.
	A. browniana H.L. Wendl.
	A. urophylla Benth.
	A. verticillata (L'Hérit.) Willd.
Myrtaceae	*Agonis flexuosa* (Sprengel) Schau.
	Hypocalymma angustifolium Endl.
Papilionaceae (Fabaceae)	*Bossiaea laidlawiana* Tovey and Morris
	B. linophylla R. Br.
Proteaceae	*Banksia grandis* Willd.
	B. seminuda (A.S. George) B. Rye
	Persoonia longifolia R. Br.
Rhamnaceae	*Trymalium ledifolium* Fenzl.
	T. spathulatum (Labill.) Ostf.
	Groundflora/shrubs
Cyperaceae	*Gahnia psittacorum* Labill.
Dennstaediaceae	*Pteridium esculentum* (G. Forster) Cockayne
Dilleniaceae	*Hibbertia amplexicaulis* Steudel
	H. hypericoides (DC.) Benth.
	H. silvestris Diels.
	H. montana Steudel
	H. obtusifolia DC.
Epacridaceae	*Leucopogon capitellatus* DC.
	L. nutens E. Pritzel
	L. verticillatus R. Br.
	Styphelia tenuiflora Lindley
Euphorbiaceae	*Phyllanthus calycinus* Labill.
Leguminosae	*Bossiaea ornata* (Lindley) Benth.
	Daviesia cordata Smith
	D. decurrens Meissner
	D. horrida Preiss ex Meissner
	D. ulicifolia Andrews
	Gastrolobium bilobum R. Br.
	G. calycinum Benth.
Liliaceae	*Dianella sp.*
Myrtaceae	*Melaleuca viminea* Lindley
Proteaceae	*Adenanthos barbigerus* Lindley
	Dryandra nivea (Labill.) R. Br.
	D. sessilis (Knight) Domin.
	Grevillea bipinnatifida R. Br.
	Hakea lissocarpha R. Br.
	H. prostrata R. Br.
	H. ruscifolia Labill.
	Synnaphea petiolaris R. Br.
Rutaceae	*Boronia littoralis* R. Br.
	B. spathulata Lindley
Santalaceae	*Leptomeria cunninghamii* Miq.
Sterculiaceae	*Lasiopetalum floribundum* Benth.
Xanthorrhoeaceae	*Xanthorrhoea australis* R. Br.
	X. gracilis Endl.
	X. preissii Endl.
Zamiaceae	*Macrozamia riedlei* (Fischer ex Gaudich.) C. Gardner

*Derived from Edgar and others (1976); Kile and Watling (1981, 1988); Kile and others (1983); Pearce and others (1986); Shearer and Tippett (1988).

saprophytic survival. Kile (1980b), Rishbeth (1972b), and Shaw (1975) have isolated *Armillaria* from stumps 40-70 years after cutting (see chapter 4). Some species also act as mycoparasites and mycotrophs, further emphasizing the ecological versatility of members of this genus.

Regarding general life history or ecological strategies (MacArthur and Wilson 1967), *Armillaria* species may be considered as relatively K-selected (the organism has a long individual lifespan and a low reproductive effort) rather than r-selected (the organism uses its energy in a short, fast growth phase accompanied by a high reproductive effort). K strategists tend towards coexistence. The Andrews and Rouse (1982) analysis of plant pathogen life histories in terms of resource allocation and the nature of the parasitic association indicates *Armillaria* species may also exhibit r-selected characteristics particularly relative to the latter. Pathogens which stress plants by reducing photosynthesis were considered relatively K-selected compared with those which induce disturbance by consuming biomass. *Armillaria* species cause host disturbance, but host debilitation is often a prolonged process; in many cases, host and pathogen may coexist for long periods. Individual species may have a broad host range, another feature associated with r-selected organisms.

Within this framework of nutritional and ecological strategies, a number of activities may be recognized for *Armillaria* species in natural forests.

Decomposer

Decomposition in forest ecosystems is effected by the integrated activity of many heterotrophic organisms, both microbial and animal (Swift 1977). The basidiomycetes play a major role in the process by breaking down complex polymeric material such as cellulose and lignin. In many forests, the role of *Armillaria* as a decomposer is its most conspicuous activity.

As a consequence of parasitic activity or disturbance such as logging, windthrow, or fire, *Armillaria* may infect large quantities of roots, stumps, and sometimes logs and other debris on the ground. In many tree species, both sapwood and heartwood may be infected, although in eucalypts infection is restricted to sapwood (Kile 1980b). *Armillaria* causes a typical white rot of infected material (see chapter 5). In the wettest forests, disintegration of the outermost tissues of stumps or logs from protracted decay may leave convoluted shapes preserved within pseudosclerotial tissue. The crunch of collapsing compartments of pseudosclerotial tissue when one walks on logs decayed by the fungus adds an audible dimension to its saprophytic activities. Frankland (1982) found the basidiomycete biomass in stumps and root material in a temperate woodland represented up to 80% of the total basidiomycete biomass on the site. The contribution of *Armillaria* species to such biomass has never been quantified, but the extensive infection observed in stumps and roots on many forest sites and the often long possession of the substrate suggest that *Armillaria* species contribute significantly to decomposition and mineral cycling within many forests. This decomposer role may also extend to the decay of timber in service, particularly under conditions of high humidity and moderate temperature (Ellis 1929, Erbisch and Harry 1979, Fassatiova and others 1974, Findlay 1951).

Mycoparasite

The diversity of resources utilized by *Armillaria* species is illustrated by the parasitism of *A. mellea* on the agaric *Entoloma arbortivum* (Berk. & Curt.) Donk (Watling 1974). Rhizomorphs invade the developing basidiomes of *E. arbortivum*, and the subsequent mycelial development induces aberrant host morphology (carpophoroids). The association appears relatively common in eastern North America. Although Watling (1974) identified the species as *A. mellea*, *A. gallica* may be the most common mycoparasitic species (Watling 1987). This is the only reported example of mycoparasitism involving an *Armillaria* species. The specificity of the relationship is not understood.

Mycotrophic (Mycorrhizal) Associations

Approximately 400 species of achlorophyllous angiosperms have evolved specialized mycotrophic root systems with basidiomycetes (Furman and Trappe 1971). These fungal associations appear necessary for the development and reproduction of the hosts. Species of *Armillaria* have been identified as associates in several achlorophyllous taxa in the Orchidaceae [*Gastrodia elata* Bl. (Kusano 1911); *G. cunninghamii* Hook.f. (Campbell 1962); *Galeola septentrionalis* Reichb.f. (Hamada 1939, 1940; Sagara and Takayama 1978)] and the Pyrolaceae [*Monotropa uniflora* L. (Campbell 1971)]. Most authors have identified the species as *A. mellea*, but recent studies have shown that in Japan *A. mellea*, *A. cepistipes*, *A. gallica*, *A. tabescens*, and possibly *A. borealis* are associated with *G. septentrionalis* (Terashita and Chuman 1987, 1989).

These associations cannot be considered as typical mycorrhizal relationships because the achlorophyllous host plant parasitizes the fungal associate for carbon compounds and nutrients which the fungi obtain from external sources (Björkman 1960, Furman and Trappe 1971, Harley 1969, Kusano 1911, Malins Smith 1952,

Zhuang and others 1983). In some cases, the mycotrophic associate is shared by the roots of the achlorophyllous angiosperm and those of a photosynthesising plant, allowing the former to indirectly parasitize the latter via a connecting bridge of mycelium or rhizomorphs in the case of *Armillaria* (Campbell 1962, Kusano 1911). Such a tripartite arrangement has been termed epiparasitism (Björkman 1960). While the mycelia involved may not depend on the host for survival, they probably derive some benefits from it. Gogala (1973) found cytokinins from *Monotropa hypopitys* L. stimulated mycelial growth of *A. mellea* and three other macromycetes.

Kusano (1911) demonstrated a well differentiated structural relationship between *Gastrodia elata* and *A. mellea* with both ecto- and endotrophic mycorrhizal features. He observed a balanced antagonism between host and symbiont in the cortical layers of the orchid tuber which involved killing of host cells by infecting hyphae or vice versa, survival of infecting hyphae in living host cells, and histochemical or cytological changes in the cells of host and fungus (see also Liu 1982, Zhang and Dong 1986, Zhang and Li 1980). The mycorrhizal hyphae showed little structural modification compared with rhizomorphic hyphae, and as the fungus could be parasitic on the orchid in some circumstances, Kusano (1911) ranked *A. mellea* as a primitive symbiont. In *G. septentrionalis*, the roots are infected by hyphae and partially differentiated rhizomorphs, and the host obtains nutrients through the digestion of hyphal coils (Hamada 1939). The structural relationship between *Armillaria* and *G. cunninghamii*, on the other hand, parallels that found in both *G. elata* and *G. septentrionalis* (Campbell 1962). The structural and cytological relationships between *M. uniflora* and *A. mellea* (Campbell 1971) have not been investigated.

Armillaria species are not known to form mycorrhizal relationships with photosynthesizing plants. Mejstřík (1969) failed to synthesize mycorrhizae between *A. mellea* and seedlings of Scots pine or Norway spruce in axenic culture.

Necrotrophic Plant Pathogen

The economic significance of *Armillaria* derives from its role as a parasite of woody plants. As a natural component of the mycoflora of native forests, *Armillaria* causes endemic disease, disease which is constantly present to a greater or lesser extent in a particular place, and distinguished from epidemic or sporadic disease (van der Plank 1975). The long coexistence of hosts and pathogens in natural forests favors a state of balance. However, since environmental or biological conditions do not remain constant, fluctuations in disease levels (local epidemics) will occur. Thus, disease caused by *Armillaria* species varies considerably in time and space.

Armillaria species may be considered as primary or secondary pathogens (fig. 8.3). As primary pathogens, they cause disease in healthy, vigorous plants which may range from restricted infections of the host tissues (root lesions, stem canker, butt rot) to progressive infections ultimately lethal to the host. As secondary pathogens, *Armillaria* species are opportunists that infect and kill trees which have been weakened by stress factors — the role of *Armillaria* species in dieback and decline diseases in natural forests.

Armillaria Species as Primary Pathogens

The ability of *Armillaria* species to act as primary pathogens in native forest communities has received less attention than their role as pathogens in plantations established on former native forest sites and as secondary pathogens in dieback and decline diseases. Two points may explain this. First, few studies have focused on the ecology of *Armillaria* in natural forests not suffering from lethal *Armillaria* disease. Second, primary disease is not necessarily lethal or to a large degree visible. As fig. 8.3 indicates, primary disease is a continuum from minor root infection to major progressive and often lethal infection; the distinction between various disease categories may at times be somewhat arbitrary.

Non-Lethal Primary Disease: Root Lesions, Cankers, Butt Rot

Armillaria appears to be abundant and widely distributed in many forests and apparently causes little disease (Boyce 1961, Peace 1962). Besides colonizing dead

Primary disease

(i) Root lesions, or root rot, basal cankers, butt rot.

(ii) Killing of natural regeneration, mortality decreasing with stand age.

(iii) Killing of trees of all ages and sizes singly or in patches throughout the life of the stand.

Secondary disease

(iv) Pre-existing or new infections kill trees weakened by stress either singly or on a stand-wide basis.

FIGURE 8.3 — The nature of disease caused by *Armillaria* species in native forests.

stumps and roots from which they may ramify through the soil, rhizomorphs may also epiphytically associate with living root systems. The fungus can also be a minor but active primary root and butt parasite. A relatively stable balance exists between host resistance and *Armillaria* pathogenicity such that, in the absence of stress, minor infections appear to have little effect on tree or forest health.

Excavation and systematic examination of tree root systems are difficult but instructive. A number of such studies in various places illustrate the common occurrence of *Armillaria* in many forests. Incidence of infected root systems in natural stands of Jack, red, and eastern white pines in the United States varied across sites from 0%-100% depending on species, stand density, and age, and was independent of tree suppression or injury (Christiansen 1938). In the Kenya Highlands, Gibson and Goodchild (1960) showed that 30% of trees surveyed in apparently healthy natural forests had epiphytic rhizomorphs or root infections. Swift (1972) found less infection in Rhodesian woodlands. In Tasmania's wet sclerophyll eucalypt forest, 74% of 300 partially excavated messmate stringybark and mountain ash had epiphytic rhizomorphs or localized root lesions (Kile 1980b). Depending on tree species, 20%-60% of healthy conifers in the northern Rocky Mountains had epiphytic rhizomorphs (McDonald and others 1987b). In Ontario, *Armillaria* root infection of black and white spruce and balsam fir varied from 31%-42%, and was influenced by tree age, soil type, and moisture supply (Whitney 1978b, Whitney and others 1974). The frequent colonization of logging stumps in the forests of southeastern Alaska also indicates a widely dispersed, indigenous *Armillaria* population infecting both stumps and, occasionally, living trees (Shaw 1981b, 1989c).

Armillaria root infection in healthy forests is limited by the hosts. Infections may be localized by resinous lesions and sapwood discoloration in conifers (Buckland 1953; Shaw 1975, 1980; Tippett and Shigo 1981) while in hardwood roots, sapwood discoloration, callus development, or kino formation (eucalypts) may occur (Kile 1980b, 1981; Shearer and Tippett 1988). Successful root infection may result in basal cankers in both hardwoods and conifers (Kile 1981, Koenigs 1969, Pearce and others 1986, Shearer and Tippett 1988) or internal (butt) decay of the stem. These host reactions are described more fully in chapters 5 and 7.

Butt rot caused by *Armillaria* is considered here as primary parasitism because it occurs in living hosts, because although most damage occurs in the heartwood entry may be gained via living root tissue, and because decay within the stem may extend outwards into the inner sapwood. Butt rot reduces wood quality and merchantable volume, and renders trees hazardous through susceptibility to stem breakage. Both coniferous and hardwood species are affected. Virtually all reports refer to *A. mellea* as the causal agent, but undoubtedly several indigenous species cause butt rot in various forests. These include *A. borealis, A. cepistipes, A. gallica,* and *A. ostoyae* in the northern hemisphere (Piri and others 1990, Rishbeth 1982) and *A. novae-zelandiae* and *A. hinnulea* in the southern hemisphere (Hood and others 1989, Kile 1980b). Records of Armillaria butt rot are summarized in table 8.2.

Stem rots have been most studied in boreal forests, particularly in North America and Scandinavia where they are considered the major cause of disease loss. The incidence of such rot varies within and among host species as determined by tree age, growth rate, stand history, and site factors (Wagener and Davidson 1954, Whitney and others 1983). *Armillaria* has frequently been noted to cause butt rot in these forests, but the subsequent direct economic loss is generally considered minor. This has been attributed to the relatively low incidence of infection and the limited extension of decay above ground level — usually less than 0.50-0.75 m, even after prolonged infection. Mechanical harvesters which shear trees close to ground level may increase the commercial significance of butt rots (Basham 1973). Armillaria butt rot has been reported to reduce pulp yields in both conifers and hardwoods (Björkman and others 1964).

Early reports of *Armillaria* causing butt rot of conifers include those of Meinecke (1916) on white fir in Oregon, and Faull (1919) and McCallum (1928) on balsam fir in eastern Canada (table 8.2). Basham and others (1953) found that while *Armillaria* sp. was isolated as frequently from butt rotted balsam fir in Ontario as *Poria subacida* (Peck) Sacc., it was of much less economic importance than the latter as infections seldom extended more than 0.6 m above ground level. In Norway spruce, volume losses to Armillaria butt rot were typically less than 10%-15% of total decay volume (table 8.2).

Armillaria butt rot of hardwoods has been recorded for species in 15 genera (table 8.2). While decay may extend further above ground in some species than in conifers (Nordin 1954, Rishbeth 1982), volume losses to butt rot remain minor. Basham (1958) found Armillaria butt rot was responsible for 8% of the total merchantable volume loss in quaking aspen in Ontario, while in Alberta it caused less than 2% loss in quaking aspen and balsam poplar (Thomas and others 1960). Greater loss was recorded for sugar maple in Ontario where Armillaria butt rot accounted for 24% of total decay volume, and infections extended an average 2 m or more above ground level depending on tree age (Nordin 1954).

TABLE 8.2 — Occurrence of Armillaria butt rot in conifers and hardwoods*.

Host species	Country or region	Importance	Reference
Conifers			
Abies amabilis (Dougl.) Forb.	British Columbia	<0.4% DV	Bier and others (1948)
	British Columbia	4.7% DV	Buckland and others (1949)
	British Columbia	0.1% DV	Foster and others (1958)
A. balsamea (L.) Mill.	Ontario	M	Faull (1919)
	Quebec	M	McCallum (1928)
	North-eastern USA	M	Spaulding & Hansbrough (1944)
	Eastern N.America	7.9% F	Basham and others (1953)
	Quebec	2.3% F	Smerlis (1961)
	Ontario	10.9% F	Basham & Morawski (1964)
	New Hampshire	M	Rizzo & Harrington (1988a)
	Eastern Canada	M	Davidson (1957)
A. concolor (Gard and Glend.) Lindl. ex Hildebr.	Oregon	M	Meinecke (1916)
A. grandis (Dougl.) Lindl.	Idaho	8.3% F	Hudson (1972)
	Idaho	14% F	Maloy & Gross (1963)
A. lasiocarpa Nutt.	British Columbia	<0.4% DV	Bier and others (1948)
	British Columbia	M	Smith & Craig (1970)
A. lasiocarpa var. arizonica (Merr.) Lem.	Arizona and New Mexico	9% F	Hinds and others (1983)
Dacrydium spp.	New Zealand	M	Birch (1937) Gilmour (1954, 1966) Hood and others (1989)
Larix decidua Mill.	United Kingdom	M	Peace (1938)
	United Kingdom	2.8% F	Greig (1962)
Phyllocladus aspleniifolius (Labill.) Hook.f.	Tasmania	M	Kile (1980b)
P. alpinus Hook f.	New Zealand	M	Gilmour (1966)
Picea abies (L.) Karst.	United Kingdom	M	Peace (1938)
	Sweden	8% DV	Rattsjö & Rennerfelt (1955)
	Sweden	M	Käärik & Rennerfelt (1957)
	Sweden	15% F	Molin & Rennerfelt (1959)
	Denmark	M	Yde-Anderson (1958)
	Denmark	10.3% F	Yde-Anderson (1959)
	United Kingdom	21% F	Greig (1962)
	Sweden	24.3% F (data combined with *P.sylvestris*)	Björkman and others (1964) [see also references in Hintikka (1974)]
	Fed. Rep. Germany	30% F	Dimitri (1966)
	Fed. Rep. Germany	6% F	Kató (1967a)
	Fed. Rep. Germany	12.7% F	Schönhar (1969)
	Fed. Rep. Germany	11% F	Zycha (1970)
	Finland	5% F	Kallio & Norokorpi (1972)
	Czechoslovakia	10-15% F	Malek (1973)
	Fed. Rep. Germany	M	von Pechmann and others (1973)
	Finland	M	Hintikka (1974)
	Finland	16% F	Kallio & Tamminen (1974)
	Norway	3.5% F	Enerstvedt & Venn (1979)
	Finland	2.4% F	Norokorpi (1979)
	Finland	<8% F	Hallaksela (1984)
	Sweden	<10% F	Stenlid & Wasterlund (1986)
P. glauca (Moench.) Voss	Eastern Canada	M	Davidson & Redmond (1957)
P. mariana (Mill.) BSP	Eastern Canada	M	Faull (1919)
		0.4% F	Basham & Morawski (1964)
P. rubens Sarg.	Eastern Canada	M	Davidson & Redmond (1957)
	New Hampshire	M	Rizzo & Harrington (1988a)
	Ontario	M	Basham (1973)
P. sitchensis (Bong.) Carr.	United Kingdom	M	Peace (1938)
	British Columbia	M	Bier and others (1946)
	United Kingdom	15.8% F	Greig (1962)

TABLE 8.2 — (Continued)

Host species	Country or region	Importance	Reference
Pinus sylvestris L.	United Kingdom	M	Peace (1938)
	Sweden	24.3% F (data combined with *P. abies*)	Björkman and others (1964)
Podocarpus spp.	New Zealand	M	Gilmour (1954, 1966)
Prumnopitys taxifolia (D. Don) Laubenf.	New Zealand	M	Hood and others (1989)
Thuja occidentalis L.	Ontario	M	Faull (1919)
T. plicata D. Don	United Kingdom	M	Peace (1938)
	British Columbia	M	Buckland (1946)
	United Kingdom	M	Gladman & Low (1963)
Tsuga canadensis (L.) Carr.	Ontario	2.9% F	Basham & Morawski (1964)
T. heterophylla (Raf.) Sarg.	United Kingdom	M	Peace (1938)
	British Columbia	6.4% DV	Buckland and others (1949)
	United Kingdom	M	Gladman & Low (1963)
	Oregon & Washington	7.4% DV	Goheen and others (1980)
	British Columbia	2.6% DV	Foster and others (1958)
Hardwoods			
Acacia dealbata Link.	Tasmania	M	Kile (1980b)
A. melanoxylon R. Br.	Tasmania	M	Kile (1980b)
Acer saccharum Marsh.	Ontario	24.3% DV	Nordin (1954)
	Ontario	20.3% F	Basham & Morawski (1964)
Betula alleghaniensis Britt.	Ontario	10.8% F	Basham & Morawski (1964)
B. pubescens Ehrh.	Sweden	6.0% F	Björkman and others (1964)
Castanopsis sp.	Papua New Guinea	M	Arentz & Simpson (1989)
Fagus grandifolia Ehrh.	Ontario	23.8% F	Basham & Morawski (1964)
Fraxinus nigra Marsh.	Ontario	54.5% F (small sample)	Basham & Morawski (1964)
Leptospermum lanigerum Sm.	Tasmania	M	Kile (1980b)
Liriodendron tulipifera L.	Eastern USA	M	Hepting & Hedgcock (1937)
	West Virginia	10% F	Byler & True (1966)
	West Virginia	9% F	Ginns & True (1967)
Lithocarpus spp.	Papua New Guinea	M	Arentz & Simpson (1989)
Nothofagus cunninghamii (Hook.f.) Oerst.	Tasmania	M	Kile (1980b)
Nothofagus spp.	New Zealand	M	Birch (1937)
Nothofagus spp.	Papua New Guinea	M	Arentz & Simpson (1989)
Ostrya virginiana (Mill.) K. Koch	Ontario	9.4% F	Basham & Morawski (1964)
Phebalium squameum (Labill.) Druce	Tasmania	M	Kile (1980b)
Populus balsamifera L.	Alberta	1.6% DV	Thomas and others (1960)
P. tremula L.	Sweden	>10% F	Eklund & Wennmark (1925)
	Sweden	5.5% F	Björkman and others (1964)
P. tremuloides Michx.	Minnesota	M	Schmitz & Jackson (1927)
	Ontario	27% F	Basham (1958) see also Black (1951)
	Alberta	0.9% DV	Thomas and others (1960)
	Ontario	9.2% F	Basham & Morawski (1964)
	Colorado	0.5% DV	Hinds and Wengert (1977)
	Quebec	M	Laflamme & Lortie (1973)
P. trichocarpa Torr. & Gray	British Columbia	3.1% DV	Thomas & Podmore (1953)
Quercus spp.	Eastern USA	M	Hepting & Hedgcock (1937)
	Eastern USA	10% F	Roth & Sleeth (1939)
Tilia americana L.	Ontario	54.5% F (small sample)	Basham & Morawski (1964)

* Covers records from both natural forests and plantations (Chapter 9), although the latter are restricted to European records for *Picea abies, P. sitchensis, Thuja plicata, Tsuga heterophylla, Larix decidua.*

M = minor if recorded as such or effects not quantified but appear to be so on the evidence presented and in the sense of causing little loss of merchantable timber volume.

% Frequency = incidence of infection in trees assessed or percentage of identified infections.

% DV = percentage of the total decay volume recorded attributed to *Armillaria* species.

Few observations in natural forests document the specific site and stand factors which might affect the incidence and severity of Armillaria butt rot. This reflects the relatively minor contribution of *Armillaria* to butt rot losses, being treated usually as incidental to those of more destructive stem-rot organisms. Birch (1937) considered overstocking may contribute to the high incidence of butt rot in silver beech pole stands in New Zealand. Basham and others (1953) noted that decay caused by *Armillaria* was more frequent in stands on poorly drained sites than in stands on relatively well drained slopes with more hardwoods in the stands.

Most butt rots are believed to develop via root infections. Basham (1958) suggested that in quaking aspen wind stress and frost heave could facilitate the entry of butt rot organisms, with occasional entry through basal wounds. Nordin (1954) found that frost cracks could provide entry points in sugar maple.

Armillaria butt rot may also occur in the same tree with other decay organisms such as *Heterobasidion annosum* (Fr.) Bref. (Kallio and Norokorpi 1972, Molin and Rennerfelt 1959), or *Armillaria* infection may allow host entry for other decay organisms such as *Phaeolus schweinitzii* (Barrett 1970, Barrett and Greig 1984, see chapter 5).

The general effects of butt rot on host growth rates and longevity are poorly understood (Wagener and Davidson 1954) though information on the relative susceptibility of some species to Armillaria butt rot has been obtained under plantation conditions (Gladman and Low 1963, Greig 1962). However, these findings may need to be interpreted in relation to the *Armillaria* species involved.

Lethal Primary Disease

Armillaria kills trees in natural coniferous and hardwood forests in different spatial and temporal patterns and with ecological and economic effects of varying significance. As noted earlier, it is part of the continuum of primary disease effects (fig. 8.3). Four such primary disease syndromes may be recognized around the world: (1) Armillaria root disease in boreal forests and western North American coastal conifer forests; (2) ring disease of mountain pine in France; (3) root rot of mixed-species conifer forests in western North America; and (4) root rot of dry sclerophyll eucalypt forests in Australia. An additional and historically interesting report of possible primary parasitism, but for which no further information is available, is that of Geschwind (1920),who observed mortality of conifers when common beech was selectively logged from mixed forest in Bosnia and Herzegovina (Yugoslavia).

The first three diseases involve mainly coniferous species and a common *Armillaria* species, *A. ostoyae*, although other *Armillaria* species may be pathogenic in boreal and mixed-species coniferous forests. On a qualitative basis the last three appear to be the most significant diseases for their impact on stand structure and progressive disease development. Other common features of these latter three diseases include their occurrence in relatively drier environments, the discontinuous distribution of the pathogens within the affected forests, the apparent greater pathogenicity of the causal *Armillaria* species compared with the same species or different species in wetter forest environments, and the apparent intensification of disease development following harvesting operations.

Root Rot in Boreal Forests and Western North American Coastal Coniferous Forests

Mortality of single or small groups of seedlings or saplings occurs early in stand development of naturally regenerated, moist coniferous forests in North America and northern Europe-Scandinavia (Baranyay and Stevenson 1964; Bourchier 1954; Buckland 1953; Hintikka 1974; Mallet and Hiratsuka 1985; Morrison 1981; Whitney 1978b, 1988a; Whitney and Myren 1978; Whitney and others 1974). Mortality typically commences soon after stand establishment, reaches a maximum at age 10-20 years, and then decreases in frequency, possibly as the food base declines and host tolerance increases. Effects on overall stocking are usually minimal, although the disease pattern may vary in some regions with limited mortality occurring through the rotation. Trees may survive with root and butt infections (Morrison and others 1985a, Whitney 1988a, Whitney and Myren 1978, Whitney and others 1974). Disease expression may sometimes be associated with stress (Buckland 1953, Baranyay and Stevenson 1964).

A number of *Armillaria* species occur in affected forests, although most disease appears coincident with the presence of *A. ostoyae* (Dumas 1988, Mallet 1989, Morrison and others 1985a, Whitney 1988a). Beyond the natural range of this species or where its distribution is limited, disease is less prominent. Thus in southeastern Alaska forests, where less pathogenic species such as *A. sinapina* may be widely distributed, little killing is evident in regeneration stands (Shaw 1981b, Shaw and Loopstra 1988). *Armillaria borealis* and *A. cepistipes* cause minor mortality and butt rot in Finnish forests (Korhonen 1978, Piri and others 1990).

Ring Disease of Mountain Pine

In relatively undisturbed 120- to 150-year-old mountain pine forests at 1,600-2,200 m in the eastern Pyrénées, mortality from *A. ostoyae* is extensive and chronic (Durrieu and others 1981, 1985; Durrieu and Chaumeton 1988). Killing may be diffuse but most

characteristically occurs in scattered but clearly delimited rings with a marginal zone of dying and dead trees (fig. 8.4). Ring diameter may reach more than 120 m and may expand 1 m per year. Historical ring development, followed on aerial photographs taken over a 36-year period (Durrieu and others 1981), indicates some rings show intermittent development while others cease expanding and gradually disappear. Following stand opening, mountain pine begins regenerating and is only moderately susceptible to *A. ostoyae*. A successional sequence occurs in the understory/ground flora until the forest returns to a pre-disease form.

The origin of the rings, the means of pathogen spread within them, and the factors controlling their initiation are poorly understood. The affected forests occur on light-textured, shallow soils, often on steep slopes; rainfall is relatively low (600-750 mm per annum), and bark beetles may act as a stress agent (Torossian 1984). However, the long-standing and strongly patterned nature of the disease and the infection and killing of provence broom, an understory species, supports *A. ostoyae* as the primary disease cause. Durrieu and others (1985) suggested the fungus is part of the forest's natural ecology, leading to the regeneration of the dominant tree species. While the disease is most severe in the Cerdagne region of France, it also extends westwards into drier transitional forests and may also occur in other parts of the range of mountain pine (Brang 1988).

Armillaria Root Disease in Mixed Coniferous Forests of Western North America

Lethal primary disease affects hundreds of thousands of hectares of natural coniferous forests in western North America. The primary documented areas of forest management concern, where Armillaria root disease occurs most extensively and severely, are eastern Oregon and Washington, northern Idaho, western Montana, and the southern interior of British Columbia. The disease is also recognized in the central and southern Rocky Mountains (Wood 1983). In these drier, interior forests, continuing mortality in all age classes is common in many stands; understocked stands may result from multiple disease centers. Armillaria root disease has been known for many decades in these forests (Ehrlich 1939; Hubert 1918, 1931, 1950, 1953) but has received minimal attention until the mid 1970's largely because the overall impact was not appreciated.

Smith (1984) estimated the average annual volume losses to five major root diseases [*Phellinus weirii* (Murr.) Gilb., *H. annosum*, *Armillaria* spp., *Phaeolus schweinitzii*, and *Ophiostoma wageneri* (Goheen: Cobb) Harrington] throughout the western United States to be 6.7 million m^3, or 18% of the total annual mortality. While the proportion of this loss due to Armillaria root disease cannot be determined, local severity has been evaluated. Shaw and others (1976a) found volume loss to *Armillaria* in a ponderosa pine stand in south-central Washington to have increased from 9 m^3 per ha in 1957 to 24 m^3 per ha in 1971. In a mixed-conifer stand in southern Oregon, Filip (1977) found 7% of trees comprising 32% of the standing volume were infected with or killed by *Armillaria*. Filip and Goheen (1982, 1984) found annual mortality of more than 3 m^3 per ha in other situations. In Montana, a root disease patch in a Douglas-fir stand contained 82% less timber volume per 0.4 ha than the adjacent healthy stand (Byler unpubl.). In British Columbia's interior cedar-hemlock zone, annual timber losses caused by Armillaria root

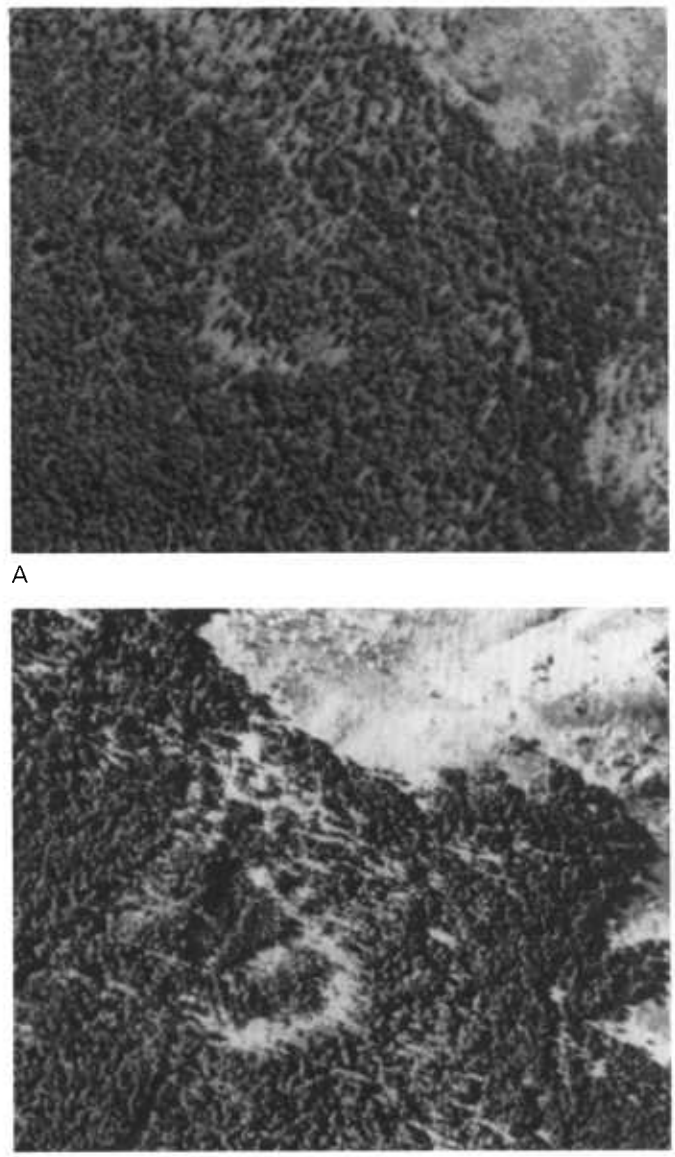


FIGURE 8.4—Development of ring disease in mountain pine forest, Pyrénées, France. A. Photographed in 1942. B. Same area photographed in 1962. (G. Durrieu)

disease were estimated to be 105,000 m^3 (Taylor 1986). Volume growth of Douglas-fir infected by *A. ostoyae* in four stands in southeastern British Columbia decreased significantly as disease severity, measured by basal resinosus, increased from infected stem bases (Bloomberg and Morrison 1989).

Armillaria was also recognized as a major cause of stand damage in other ground and aerial surveys which have recorded incidence and area of root disease centers (Williams and Leaphart 1978). James and others (1984) estimated active root disease centers, mainly attributed to *Armillaria* and *Phellinus weirii*, occupied almost 32,000 ha (about 1% of the total commercial forest land) of seven national forests in the northern Rocky Mountains. A detailed study of one of those forests, the Lolo, found 123,255 ha (18.8% of the total forest) were diseased, of which 8,011 ha (1.2%) were unstocked patches (Byler and others 1990).

Besides timber loss and the creation of unproductive areas through chronic infection, particularly where susceptible hosts are climax (McDonald and others 1987a), Armillaria root disease may change species composition, create hazardous trees in recreation forests, and affect the choice of silvicultural system.

Armillaria ostoyae (NABS I[1]) (Morrison and others 1985a, Wargo and Shaw 1985) and possibly NABS X (McDonald unpubl.) are pathogenic on conifers in these interior western forests, although *A. ostoyae* is considered the most widespread and aggressive. Additional taxonomic or biological species known to be present in western North America are *A. sinapina* (NABS V), *A. gallica* (NABS VII), NABS XI *(A. cepistipes?)*, and NABS IX (Anderson and Ullrich 1979, Morrison and others 1985a, Shaw and Loopstra 1988, Wargo and Shaw 1985). The latter two species have been collected infrequently. Some of these species may act as secondary pathogens.

Where they occur, *Armillaria* species have a complex interaction with about two dozen conifer species. Data on mortality rates resulting from root disease caused by *Armillaria* in different community types and geographic areas are lacking, although observations indicate Douglas-fir and true fir are the most susceptible (Hagle and Goheen 1988, Morrison 1981). Exceptions to this occur in south-central Washington where ponderosa pine is most susceptible and Douglas-fir appears tolerant, and possibly in some other areas where Engelmann spruce (McDonald and others 1987b) and western hemlock appear very susceptible (Morrison 1981). Root disease may also afflict hardwood shrubs (Adams 1974; McDonald and others 1987a,b; Morrison 1981; Shaw 1975; Tarry and Shaw 1966; Williams and Marsden 1982).

In individual stands, mortality often begins within a few years of regeneration and may continue throughout the rotation, particularly in Douglas-fir/true fir forests. For other species, such as western redcedar, mountain hemlock, western larch, western white pine, ponderosa pine, and lodgepole pine, damage tends to diminish with stand age beyond 20-30 years. Disease occurrence varies from individual infected trees (fig. 8.5) to patches (fig 8.6) of tens of hectares (Byler unpubl., Filip 1977, James and others 1984, Smith 1984, Wargo and Shaw 1985). Patches typically contain conifer regeneration, brush, or grass and have a perimeter of dead and dying trees. Rate of disease spread in a ponderosa pine stand was 1-2 m per annum (Shaw and Roth 1976), but in a Douglas-fir stand less than 0.25 m per annum (Byler unpubl.). Typical infection foci are usually occupied by 1-3 *Armillaria* genotypes (Adams 1974, McDonald and Martin 1988, Shaw and Roth 1976). The dynamics of disease within infection centers and across rotations in these mixed-conifer forests is discussed further in chapter 10.

Armillaria frequently causes damage concomitant with other root-rot pathogens of mixed-conifer forests, particularly with *Phellinus weirii* (Filip and Goheen 1984, Goheen and Filip 1980, James and others 1984, Miller and Partridge 1973. Williams and Leaphart 1978), but also with *O. wageneri* (Cobb and others 1974) and *H. annosum* (F. Cobb pers. comm.). *Armillaria* may be active on the same site or in the same root system as other pathogens. Hansen and Goheen (1989) attributed these associations to chance and to primary-secondary relationships, but the roles have not been adequately defined.

Armillaria infection and other root diseases predispose some conifers to bark beetle infestation (Hertert and others 1975, Hinds and others 1984, James and Goheen 1981, Kulhavy and others 1984, Lane and Goheen 1979, Lessard and others 1985, Partridge and Miller 1972, Tkacz and Schmitz 1986). Armillaria root disease may be an important factor in the survival of endemic populations of some bark beetle species. Hinds and others (1984), Lessard and others (1985), and Tkacz and Schmitz (1986) associated such populations of the mountain pine beetle (*Dendroctonus ponderosae* Hopkins) with *Armillaria* infection in ponderosa and lodgepole pine stands in South Dakota and Utah, respectively. Interaction between bark beetles and Armillaria root disease is considered further in chapter 10.

Western North America is marked by complex landforms and specific associations of plant communities. Large variation in elevation, aspect, slope, altitude, and

[1]NABS (North American Biological Species) as described fully in chapters 1 and 2.

A B

FIGURE 8. 5— Armillaria root disease killing individual trees near infected stumps in a mixed-species conifer forest in western North American. A: Ponderosa pines; B. Grand fir.

FIGURE 8.6—Armillaria root disease center in virgin coniferous forest in western North America. The lowermost center covers nearly 8 ha (20 acres).

soil type has produced an elaborate mixture of forest ecosystems with widely differing levels of vulnerability to Armillaria root disease. Root disease centers have been associated with particular forest habitat types (Byler and others 1986, 1990; McDonald 1990; McDonald and others 1987a,b; Williams and Marsden 1982).

Armillaria root disease probably played an important role in forest succession and the determination of stand composition and structure on many mixed-conifer forest sites prior to European settlement (Byler 1984, Byler and others 1990, Hagle and Goheen 1988, Haig and others 1941, Shaw and Roth 1976, Wargo and Shaw 1985). *Armillaria ostoyae*, for example, accelerates succession in interior British Columbian forests especially on wetter sites. There, the pioneer species (usually Douglas-fir or lodgepole pine) is killed and the openings fill with shade-tolerant western hemlock or western redcedar after Douglas-fir, or subalpine fir after lodgepole pine. These species are not markedly less susceptible to *A. ostoyae* but appear to be more tolerant, more frequently restricting infection to root lesions and butt rot (Morrison 1981). Williams and Marsden (1982) suggested a similar role for *Armillaria* and *Phellinus weirii* in the succession on northern Idaho sites where western hemlock was climax. Disease is also evident in other forests undisturbed by human activity (Haig and others 1941, Wargo and Shaw 1985).

Armillaria Root Disease in Dry Sclerophyll Eucalypt Forests

As a primary pathogen, *A. luteobubalina* affects many eucalypt and understory species in dry sclerophyll mixed-species eucalypt forests in central Victoria, and in karri and jarrah forests in southwestern Western Australia (Kile 1981, Kile and others 1983, Pearce and others 1986, Shearer and Tippett 1988). The affected forests occur between 300 m and 1,200 m altitude on soils of variable fertility, and receive annual rainfall of 700-1,200 mm. Most have a long history of logging. Hosts in these forests include at least 81 eucalypt, understory, and ground species (table 8.1).

The evidence for the primary pathogenicity of *A. luteobubalina* includes the constant association of the fungus with disease, a pattern of contagion consistent with that for an organism dependent on a woody food base, a correlation between infection and symptom development in large trees, and pathogenicity of the fungus in pot and field inoculations of some host tree species (Kile 1981, Pearce and others 1986, Shearer and Tippett 1988).

In Victorian forests, diseased trees tend to occur in roughly circular foci although the pattern of disease development is often obscured by multiple infection, cutting, and burning (fig. 8.7). Within patches, which may range from a few trees to 1 ha or more, the disease usually shows progressive outward expansion, with more recently dead and dying trees towards the margin and older dead and often wind-thrown trees towards the center. The chronic nature of infection is apparent by the death of eucalypt or understory regeneration that was established following death or removal of the previous strata. Typically, *A. luteobubalina* or other *Armillaria* species are not found in healthy forest surrounding diseased areas. Similar disease development occurs in jarrah forest. In karri forest, the disease is most active in young stands; with increasing stand age, mortality is restricted to suppressed or subdominant trees although larger trees may be infected (Pearce and others 1986).

Young infected trees often die suddenly with a major proportion of their foliage intact. In contrast, large, mature trees generally show progressive crown dieback before eventual death. Some trees develop basal cankers from infection, which limit fungal spread and promote host survival (Kile 1981). The fungus forms few rhizomorphs in forest soils, and underground spread between hosts occurs via root contacts at an average rate of 1-1.5 m per annum (Kile 1983b).

Several thousand hectares of Australian eucalypt forest are seriously affected by the disease (Edgar and others 1976, Shearer and Tippett 1988). Edgar and others (1976) estimated mature stands with moderate to severe disease had respective sawlog increments of about one-half and two-thirds that of an average healthy stand, with growth losses of 0.3-2.0 m^3 per ha per year depending on site and disease severity. Besides these losses, scattered and small patch mortality is evident in regrowth stands. In 30-year-old regrowth messmate stringybark, with 51-75% of ground-level stem circumference infected by *A. luteobubalina*, average monthly girth increment was only 41% of that of healthy trees (Kile and others 1982).

FIGURE 8.7 — Aerial view of *Armillaria luteobubalina* root disease center in dry sclerophyll eucalypt forest, Victoria, Australia.

The wide distribution of *A. luteobubalina* in southern Australia and its intimate association with eucalypt forest communities indicate that it is indigenous. While Kile (1983a) reported evidence of its pathogenic activity in unlogged eucalypt forest, the greatest incidence and severity of disease has been observed in selectively logged forests (Edgar and others 1976). Strong relationships exist between infected stumps and disease incidence (Edgar and others 1976, Kellas and others 1987, Kile 1981, Pearce and others 1986). Though disease is endemic, logging apparently alters the balance between host and pathogen toward more severe local epidemics.

Armillaria Species as Secondary Pathogens

Biotic or abiotic stress of natural forests or individual trees (see chapter 7) within them may transform indigenous *Armillaria* species into vigorous secondary pathogens. This phenomenon is most notable in forests where, prior to stress onset, disease is restricted to epi-

phytic associations, root lesions, and butt rot. This secondary role has been recognized since early this century (Nechleba 1915; see also reviews by Day 1929 and Twarowski and Twarowska 1959) and has often dominated views of the pathogenic behavior of *Armillaria* species (Day 1929, Gremmen 1976).

Virtually all historical reports of secondary pathogenesis refer to *A. mellea*, but many other species also act in this manner. The identity and relative importance of species of different pathogenicity in broadscale secondary diseases such as those shown in table 8.3 therefore require reappraisal.

Although forest diebacks and declines are episodic diseases of varying etiology, all share a causal complex that begins when tissues of healthy trees are altered or predisposed by stress and culminates when those tissues are invaded and killed by facultative parasites (Houston 1973, 1982, 1984, 1987). Because infections by weakly pathogenic organisms are unsuccessful or restricted in the absence of stress, and because in the absence of these organisms trees usually recover with the abatement of stress, organisms of secondary action such as *Armillaria* species are an integral component of the disease syndromes. This does not imply, however, that stress alone cannot kill trees (Houston 1987).

Stress factors include insect damage, primary pathogens, drought, waterlogging, fire, temperature extremes, air pollution, or silvicultural treatments. These stresses may be either protracted or relatively ephemeral, and they may occur months or even years prior to eventual tree mortality. Not all stresses enhance pathogenic activity, however, and some air pollutants probably have an adverse effect on the fungus itself, restricting its ability to take advantage of weakened hosts (Singh and Sidhu 1989).

The prominent role of *Armillaria* species in diseases such as those shown in table 8.3 results from their extensive natural distribution in the stress-affected forests and their primary infection of or epiphytic presence on many root systems prior to the advent of stress. The fungus is thereby able to take advantage of changed circumstances to spread quickly from existing infections or establish new ones. For example, regarding dieback of regrowth messmate stringybark and mountain ash in Tasmania, *A. hinnulea* and/or *A. novae-zelandiae* usually infected a large proportion of each tree's root system at the time of death. Excavations, however, indicated that in healthy forest at least 70% of trees had minor root infections or epiphytic rhizomorphs (Kile 1980b, Kile and Watling 1983). In this and many other diebacks and declines, *Armillaria* is probably responsible for the ultimate death of many trees.

Unlike lethal primary disease caused by *Armillaria* species, where dead and dying trees are usually clustered in expanding foci, the pattern of mortality in dieback and decline diseases is typically more variable, ranging from essentially random to more site or topographically related patterns. *Armillaria* infection is less readily associated with identifiable food bases. The distribution of different *Armillaria* species may explain variations in infection and subsequent patterns of mortality, because species of different pathogenicity may invade root systems at different stages of host debilitation (Guillaumin and others 1989a). The susceptibility of individual trees or stands to infection will be mitigated by site and soil factors and tree vigor.

Experimental studies have demonstrated the increased susceptibility of various tree species to infection when stressed by defoliation, suppression, reduced light intensity, adverse soil moisture conditions, or nutrient supply (Davidson and Rishbeth 1988, Entry and others 1986, Ono 1970, Redfern 1978, Wahlstrom and Unestam 1989, Wargo 1972, Wargo and Houston 1974). Increased susceptibility is related to biochemical changes in the host induced by stress, which lowers host resistance and stimulates development of the fungus. Individual stress factors and their effects on pathogenesis by *Armillaria* are fully discussed in chapter 7.

Dispersal and Distribution

The spatial development of *Armillaria* populations in natural forests ranges from the discontinuous distribution of discrete genotypes of one species, to a mosaic of genotypes to which one or more species may contribute. Through the infection of living hosts, stumps, and roots and the proliferation of rhizomorphs, the latter situation can be equated to a continuous distribution, although even in multi-species populations individual species may have restricted occurrences. Discontinuous distributions appear more typical of temperate, Mediterranean, and tropical forests while continuous distributions are more evident, although not omni-present, in boreal and cool temperate forests. However, better quantification of spatial distributions are required in relation to both *Armillaria* species and forest type.

In boreal and temperate forests, genotypes of different *Armillaria* species have been identified and mapped using alleles of the incompatibility (mating) genes as genetic markers or by intraspecific pairings of diploid forest isolates (see chapter 2).

Dispersal and distribution occur via basidiospore infections that create new infection foci and vegetative growth that expands the local distributions of particular genotypes. Local expansion may proceed by

TABLE 8.3 — Examples of diebacks and declines in natural forests in which *Armillaria* species were recognized as important secondary pathogens.*

Disease and primary host	Major initiating stress	Location	Time frame	Reference
Alaska yellow-cedar dieback(*Chaemaecyparis nootkatensis*	Unknown	Southeast Alaska	early 1900s to present	Frear (1982) Shaw and others (1985) Hennon and others (1990)
Dieback and mortality of coniferous species	a,c	Eastern Canada	late 1960s-early 1980s	Hudak and Singh (1970) Hudak and Wells (1974) Raske and Sutton (1986)
Birch dieback *(Betula alleghanensis)*	a,c,f,g	Northeastern North America	mid 1930s-late 1950s	Spaulding and MacAloney (1931)
Oak declines (*Quercus* species)	a,c,d	1) Europe	regional occurrences during this century	Baumgarten (1912) Hen (1914) Falck (1918) Georgevitch (1926b) Day (1927a) Stolina (1954) Petrescu (1974) Guillaumin and others (1983)
	a,c,d,g	2) Midwest and eastern USA		Macaire (1984) see reviews by Staley (1965) and Houston (1987) Wargo (1977)
Ohia decline *(Meterosideros polymorpha)*	b,e	Hawaii	mid 1950s-early 1970s	Laemmlen and Bega (1974) Hodges and others (1986)
Pole blight *(Pinus monticola)*	a	Western USA British Columbia	1930s-1950s	Hubert (1950, 1953) Leaphart and others (1957)
Regrowth dieback *(Eucalyptus regnans, E. obliqua)*	a,c	Tasmania	regional occurrences early 1960s-present	Kile (1980b)
Sugar maple declines *(Acer saccharum)*	a,c,f	Eastern North America	regional occurrences 1950s-present	Houston and Kuntz (1964) Wargo and Houston (1974)

* The derivation of the table arrangement and some data from Houston (1987) is acknowledged.

Stress factors
a water deficit/high temperature
b poor drainage/water excess
c defoliation by insects
d defoliation or damage by fungi
e nutrient imbalance
f logging disturbance
g low temperature damage

rhizomorphs or mycelial growth through and between contacting root systems. The role of basidiospores as inoculum and the involvement of rhizomorphs in spread and infection are considered fully in chapter 4, and comment here is restricted to points particularly relevant to these processes in natural forests.

Basidiospores

Although the potential epidemiological importance of basidiospore infection has long been recognized (Boyce 1961, Hiley 1919, Rishbeth 1964), the evidence for its occurrence in natural forests remains circumstantial based on the detection of multiple genotypes and unique combinations of mating alleles (Kile 1983b, Korhonen 1978).

As with many other macromycetes, spore production by *Armillaria* basidiomes may be prolific and the period for potential basidiospore infection relatively long. Rishbeth (1970) recorded deposition rates of up to 1,000 viable basidiospores per dm^2 per min. close to basidiomes. Basidiospores have also been trapped from the air on screens and on freshly cut wood (Hood and Sandberg 1987, Molin and Rennerfelt 1959, Rishbeth 1970, Swift 1972). Basidiome production of some

Armillaria species may extend over several months (Fedorov and Bobko 1989, Kile and Watling 1981, Pearce and others 1986, Rishbeth 1970, Shubin 1976). Shaw (1981a) found basidiospores could remain viable on the outer bark of conifers over an Alaskan winter.

Environmental factors, particularly moisture, host, and *Armillaria* species, may influence spread and infection by basidiospores (Rishbeth 1970). In the relatively dry interior forests of western North America, few genotypes of *A. ostoyae,* and the large areas occupied by some of them, suggest limited opportunities for basidiospore infection (Shaw and Roth 1976). Similarly, in dry sclerophyll eucalypt forest in Victoria, where 36 genotypes were found in 24 ha, Kile (1983b) estimated the rate of basidiospore infection for *A. luteobubalina* could average less than one per year. In wet sclerophyll eucalypt forest in Tasmania, 46 genotypes of *A. hinnulea* were isolated in 1.1 ha, suggesting relatively frequent basidiospore infection in this forest type (Kile 1986). A situation comparable to the latter probably exists in deciduous forests in the Northeastern United States, Finnish coniferous forests, and New Zealand hardwood-podocarp forests where moisture conditions are favorable for frequent and abundant basidiome production (Anderson and Ullrich 1979, Hood and Sandberg 1987, Korhonen 1978).

Because of our poor knowledge of basidiospore infection courts and the potentially large number of factors which may influence the incidence of basidiospore infection, further experimental studies of the process are needed.

Rhizomorphs and Root Contacts

Both rhizomorphs and root contacts are important for spread and infection in natural forests. The actual contribution of rhizomorphs to these two processes probably depends on the forest environment and the characteristics of the rhizomorphs of the particular *Armillaria* species (see chapters 4 and 6), but they are not obligatory for either spread or infection.

Infection via root contact has received little attention in the past, but its efficiency in some forests suggests it could contribute to spread even in situations where rhizomorphs are present. *Armillaria luteobubalina* spreads almost exclusively by this means in Australian eucalypt forests even though it produces rhizomorphs on agar and in pot culture (Kile 1981, Morrison 1989, Pearce and others 1986, Podger and others 1978, Shearer and Tippett 1988). Rhizomorphs also are formed rarely in many tropical forests (Butler 1928; Dade 1927; Swift 1968, 1972).

The lack of rhizomorphs in native forest soils has been attributed to unfavorable physical environments for their initiation and growth (Pearce and Malajczuk 1990a, Rishbeth 1968) or to inhibitory compounds in the soil (Olembo 1972, Swift 1968). Pearce and Malajczuk (1990a) showed limited rhizomorph development of *A. luteobubalina* in karri forest soils in Western Australia related to unsuitable combinations of soil temperature and soil moisture levels for rhizomorph growth during much of the year. Whether this explanation is adequate in other forests where rhizomorphs are absent remains to be determined.

Spatial Distributions

Over time, dispersal processes contribute to varied patterns of genotype and species distribution. These patterns appear to form a continuum from the simple to the complex depending on the frequency of new infections and the number of *Armillaria* species. In coniferous forests in western North America and dry sclerophyll eucalypt forests in Victoria, relatively few genotypes of *A. ostoyae* and *A. luteobubalina*, respectively, may develop large, discrete, clones (2-3 ha or more) with individual genotypes occurring hundreds of meters apart (Adams 1974, Anderson and others 1979, Hood and Morrison 1984, Kile 1983b, Shaw and Roth 1976) (fig. 8.8). The size of some clones and the discontinuous distribution of some genotypes when compared with rates of spread is taken as evidence that the original infections may have begun decades or even several centuries previously (Kile 1983b, Shaw and Roth 1976). In contrast, *A. borealis, A. ostoyae, A. gallica, A. cepistipes, A. mellea, A. hinnulea, A. limonea,* and *A. novae-zelandiae* form relatively small clones (approx. 50-100 m maximum distance between isolates of the same genotype) in a variety of coniferous and moist temperate hardwood forests. A relatively frequent number of genotypes occurs per unit area, often in close proximity or intermingling and possibly with clones of more than one species (Hood and Sandberg 1987, Kile 1986, Korhonen 1978, McDonald and Martin 1988, Rishbeth 1978b, Thompson 1984, Ullrich and Anderson 1978).

Once established in the forest, any given genotype may persist for decades or centuries, occupying successive woody substrates as a result of parasitic and saprophytic activity. Multiple genotypes form a type of subterranean mosaic, but the physical and biochemical interaction between intra- or inter-specific genotypes has received limited investigation (Mohammed and Guillaumin 1989). These kinds of interactions could be mediated by extracellular structures such as those observed in *Postia placenta* (Fr.) M. Lars. et Lomb. (Green and others 1989), although they have not been observed in *Armillaria*.

Studies of dispersal and distribution emphasize the dynamic nature of the interaction between *Armillaria* species and natural forest ecosystems. Kile (1983b)

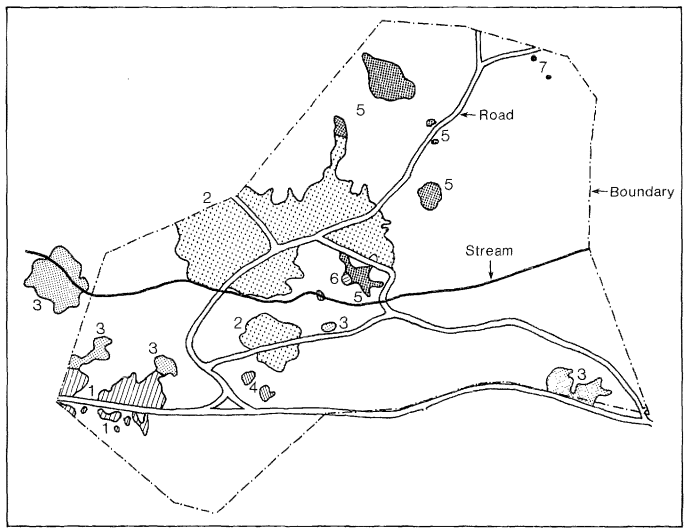


FIGURE 8.8 — Occurrence of seven genotypes of *Armillaria luteobubalina* in a eucalypt forest in the 33 ha Victoria Mill Scenic Reserve, Mount Cole State Forest, Victoria, Australia. (From Kile 1983b, courtesy Australian Journal of Botany)

considered that the major factors influencing clonal development were likely to be: the number and location of existing and new infections, the pathogenicity of the individual genotypes, their longevity in individual food bases, the stand and tree characteristics including host resistance, and perturbations within the forest such as fire and logging. Combinations of such factors over long periods could account for the limited size of some clones, the extensive or dispersed distribution of others, the presence of multiple genotypes in the same area, contraction or expansion of clone size, differing disease intensity, and predictably the loss of some genotypes from the forest.

Pathogenicity, Environment, Host Resistance, and Primary Disease Expression

As in other plant diseases, the expression of Armillaria root disease is influenced by species pathogenicity, host resistance, and environmental factors. How these factors interact relative to primary disease in natural forests is poorly understood at the present time.

A general observation can be made from the disease reports considered in this chapter: while non-lethal primary disease may be common in relatively wetter boreal and temperate forests (which may be translated into lethal secondary infection by stress), the most serious primary disease occurs in relatively drier Mediterranean or continental forests. As there is no evidence that recent introductions of pathogenic species are responsible for disease, the current situation presumably reflects the results of long-term coevolution of hosts and pathogens.

One explanation for a difference of this nature in disease epidemiology encompasses the pathogenicity of the indigenous *Armillaria* species and the environmental and biological factors which control the population of the fungus. The large food base which may develop in wet forests in mild climates is seemingly balanced by the weak pathogenicity of the resident *Armillaria* species. In harsher forest environments, stress may have selected species or genotypes of greater pathogenicity which can effectively maintain themselves in the forest community from a more limited food base. In the former forests, weak pathogenicity, wide distribution, and long survival in inoculum are the elements of mutual coexistence (a feature of K-selected organisms). In the latter forests, greater pathogenicity, discontinuous distributions, and shorter survival in inoculum achieve the same end. In neither situation is the survival of the host species threatened. When stress leads to secondary *Armillaria* infection, not all trees are killed and such events typically lead to the establishment of regeneration.

These concepts, represented in fig. 8.9, can be illustrated by Australian examples. In Tasmania's wet sclerophyll eucalypt forest, *A. hinnulea* / *A. novae-zelandiae* are almost ubiquitous on root systems as epiphytic rhizomorphs or root lesions (Kile 1980b, Kile and Watling 1983). Logging or wildfire creates a vast food base and results in a high incidence of massive root and stump infection but with virtually no mortality among the regenerating stand of eucalypts and other species. Disease is restricted to the same form as that in the pre-existing stand (Kile 1980b). In Victoria's dry sclerophyll forest, on the other hand, eucalypts and other species are killed by *A. luteobubalina* even in the absence of artificial disturbance (Kile 1981, 1983b). However, food bases are fewer and the fungus generally only survives in stumps for 15-25 years (Kile 1981).

The relative pathogenicity of different *Armillaria* species on the same host under controlled conditions can be ranked (Morrison 1989). Variable disease expression on the same host supports the view that the differences described for eucalypt forests are to a significant degree caused by interspecific differences in pathogenicity rather than by host or short-term environmental effects. Messmate stringybark is a common host in both forest types, yet is only killed where *A. luteobubalina* occurs (Kile 1980b, 1981). Direct pathogenicity comparisons of the three *Armillaria* species present in the forests have not been made on messmate stringybark, but those tests undertaken with the individual species support the view that *A. luteobubalina* is inherently more pathogenic than either *A. hinnulea* or *A. novae-*

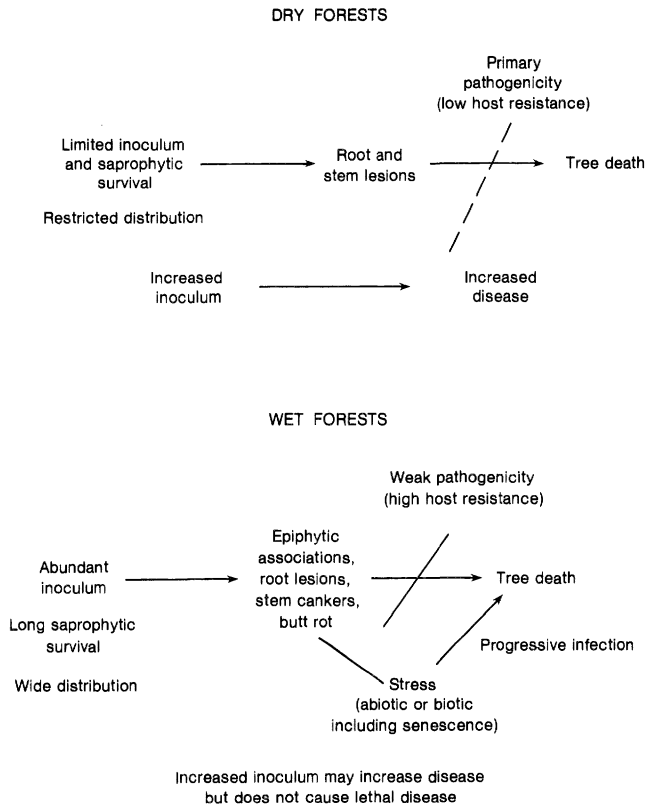


FIGURE 8.9—Conceptual models of the pathogenic behavior of *Armillaria* species in terms of two general forest environments.

zelandiae (Kile 1980b, 1981; Morrison 1989; Podger and others 1978).

Intra-regional differences in the epidemiology of disease caused by the same *Armillaria* species on the same or related hosts have been observed. In western North America, *A. ostoyae* is an aggressive pathogen on interior forest species but usually only causes minor disease on many of the same hosts in wet coastal forests (Morrison 1981, Morrison and others 1985a, Wargo and Shaw 1985). A similar situation may occur with *A. luteobubalina* in Western Australian forests. Shearer and Tippett (1988) noted that host mortality following infection was greater in the intermediate and low rainfall zones of the eastern jarrah forest than in the higher rainfall zones to the west. The fungus also appears to be less damaging in the wetter karri forest in the same region (Pearce and others 1986).

For western North America, Morrison and others (1985a) considered these differences might result from intraspecific variation in pathogenicity between coastal and interior isolates of *A. ostoyae*. McDonald and others (1987a,b) proposed that the difference in pathogenic behavior is linked to site productivity, host adaptation, or stress, with the incidence of pathogenic behavior showing a strong tendency to decrease as stand productivity increased (fig. 8.9). They further suggested that disease incidence is also greatest in host populations in transition zones between climax species. These are seen as being less adapted to the site and therefore more vulnerable through lower host resistance. Soil factors may also be influential (Shields and Hobbs 1979, Williams and Marsden 1982). Intraspecific variation in host resistance has not been investigated relative to primary disease but could help explain regional differences in intraspecific pathogenicity. Presently, there is little basis on which to judge the merits of these different hypotheses, but they lend themselves to creative experimentation.

Possible feedback mechanisms such as that proposed for mountain hemlock stands infected by *Phellinus weirii* may also operate to influence host resistance during stand development. Waring and others (1987) suggested pathogen-induced disturbance may increase nutrient and light availability following death of mature stands, and increase resistance of young trees against infection. Although Shaw and others (1988) challenged this assumption and suggested vigorously growing young trees would have more roots and thus an increased probability of infection, such controversy does not deny the possible existence of such effects.

Disease expression in natural forests is clearly a complex phenomenon. Establishing the significance of host, pathogen, and environmental moderation in disease expression is a major challenge for future research. These interactions and consequent disease expression are in turn modified by forest management practices.

Forest Management and Disease

Forest harvesting and other disturbances cause fluctuations in inoculum levels in natural forests (Kile 1980b). In forests where the major *Armillaria* disease effects are non-lethal (root lesions, minor root rot, and butt rot), these fluctuations appear to have little effect on disease epidemiology from crop to crop unless management severely stresses residual trees. Where *Armillaria* species are aggressive primary pathogens, such as in the situations already described, management practices such as logging and control of fire severity and frequency may significantly affect disease expression. These may occur directly through impact on inoculum levels or interactions with species composition and stand structure. Much of the available information is observational, however, and little experimental study has been done on the effect of management practice on disease levels.

Selective logging in old-growth or mature-regrowth stands may intensify disease development in the re-

sidual stand. Damage is dependent on stand age and species and is usually attributed to increased inoculum, but physiological stress from exposure in retained trees could also be of some importance in increased tree susceptibility (see chapter 7). Logging old-growth ponderosa pine in southern Washington State led to striking disease development in young trees, with zones of dead and dying seedlings and saplings surrounding the infected old-growth stumps and eventually leading to the creation of an open area (Shaw 1975, Shaw and others 1976a). Harvesting, particularly selective logging, has also led to inoculum buildup on many species of conifer stumps in other western North American forests (Byler and others 1990, Filip and Goheen 1982, Hagle and Goheen 1988). Severe Armillaria root disease in dry sclerophyll eucalypt forest in Victoria, Australia was associated with repeated (approximately 10-year) selection cutting of the larger trees (Edgar and others 1976). Subsequently, Kellas and others (1987) showed that cutting intensity per se did not affect disease incidence, but that frequency of cutting within infected forests is probably the critical factor promoting disease development. Regular creation of stumps increased inoculum levels and the probability of residual trees being in close proximity to inoculum, thereby altering the balance in favor of the pathogen. Unless *A. luteobubalina* can access and infect stumps within 3-4 years of cutting, it is excluded from colonization by other microorganisms (Kile 1981). Clearfelling in disease patches could therefore be a better management practice by reducing the number of stumps infected and the disease level in the subsequent crop.

Partial cutting practices may make stands more susceptible to disease through changes in species composition. Selective logging in mixed-conifer forests in western North America, particularly those where species of pine and larch predominate, can favor regeneration of the more root-disease-susceptible Douglas-fir and true fir (Byler and others 1990, Filip and Goheen 1982, Hadfield 1984, Hagle and Goheen 1988, Shaw and others 1976a). Such changes may not always be adverse, however. Shelterwood cutting in dry sclerophyll eucalypt forest favors regeneration by broad-leaved and narrow-leaved peppermint as opposed to messmate stringybark, the commercially preferred species (Kellas and others 1987). The former species have a similar susceptibility to *A. luteobubalina*.

It may be feasible to space or commercially thin young, even-aged regrowth stands without increasing levels of Armillaria root disease if small stumps do not provide a sufficient food base to establish new disease foci, or tree vigor is enhanced sufficiently to resist root disease. Filip and others (1989) and Johnson and Thompson (1975) found no adverse effects on stocking 20 years after thinning in a young ponderosa pine stand, and although Koenigs (1969) found thinning in an 80-year-old released understory stand of western redcedar increased root rot, disease was also apparently influenced by other stress factors. Precommercial thinning is generally not recommended in *Armillaria*-infected Douglas-fir regeneration (Morrison 1981). Further experimental studies on effects of spacing and thinning in relation to species composition appear necessary, however, before firm conclusions can be made.

Fire management can influence the susceptibility of forest stands to disease. Fire may directly affect *Armillaria* activity in forests through destruction of inoculum or indirectly through stress effects on the fungal mycelium which lead to natural biocontrol (Reaves and others 1990). Although a reduction in disease has not been demonstrated, fire frequency and intensity may also be a major determinant of the susceptibility of stands to disease through its influence on tree vigor, species composition, and stand structure.

No known studies quantify the effect of fire on inoculum quantity and viability, although Hood and Sandberg (1989) reported reduced isolation success from rhizomorphs on a clearcut native forest site after burning. General observations suggest a significant direct effect on inoculum levels is only achieved by high-intensity fire which burns or chars stumps and major buttress and lateral roots (Kile 1980b, 1981). Even then it is likely a significant proportion of below-ground inoculum will escape direct effects.

Munnecke and others (1976) showed that heating severely weakens the vitality of *Armillaria* mycelium, rendering it susceptible to parasitism by *Trichoderma viride* Pers.:Fr. and other soil-inhabiting fungi. Similar effects may operate to reduce inoculum in burned forests. Reaves and others (1990) found isolates of *Trichoderma* species from burned and non-burned soils beneath a ponderosa pine forest in Oregon were antagonistic to *A. ostoyae*, reducing colony growth and rhizomorph formation in culture. Isolates from burned soils were more antagonistic than those from non-burned soils as fire favored the growth of more antagonistic *Trichoderma* species. In the same situation, ash leachates inhibited growth of *A. ostoyae* in vitro while having a positive effect on *Trichoderma* (Reaves and others 1984, 1990). While appropriate use of fire may be effective in elevating populations of *Trichoderma* that are antagonistic to *Armillaria*, the mechanism, extent, and persistence of such effects need clarification.

In forest types in which burning has been a determinant of species composition and stand characteristics, fire suppression or exclusion may interact with silvicultural management to promote Armillaria root disease by allowing regeneration of species which are more

susceptible to *Armillaria* (Byler 1984, Byler and others 1990, Filip and Goheen 1982, Hagle and Goheen 1988, Shaw and others 1976a). Fire control associated with selective logging in drier coniferous forests in western North America has favored regeneration and overstocking of Douglas-fir and true fir in stands formerly composed predominantly of ponderosa and white pine and western larch, species apparently less susceptible or more tolerant to root disease. A need exists for more careful consideration of long-term ecological effects induced by various stand treatments on Armillaria root disease. A better understanding of such effects could lead to refinement of silvicultural methods.

While not a direct management effect, introduction of white pine blister rust (*Cronartium ribicola* J.C. Fisch.) into the northern Rocky Mountains of western North America has likely enhanced *Armillaria*-caused mortality (Byler and others 1990). The rust epidemic emulated a partial cut by killing large numbers of pole-sized and larger western white pines in many individual stands. It also modified succession in new stands by reducing or eliminating western white pine regeneration. A dramatic shift in species composition of many stands from the tolerant western white pine to susceptible Douglas-fir and grand fir was one result. The rapid killing of larger trees probably contributed to inoculum buildup as well. A related problem was the application of many western white pine salvage cuts due solely to the threat of *C. ribicola*.

Apart from direct management effects on quantity of inoculum or species composition, practices which severely stress plants may also increase disease. *Armillaria* species are thus a potential hazard for intensively managed coppice forests. Incidence and extent of root rot increased with shortened rotation in quaking aspen and bigtooth aspen sucker stands in Ontario and Wisconsin, and sucker numbers declined as a greater proportion of stumps were invaded by the fungus in successive rotations (Stanosz and Patton 1987a,b; Stiell and Berry 1986). Chronic low-level primary disease in the natural forest was transformed into more progressive secondary infection of physiologically stressed stumps and root systems by this type of management system.

Conclusions

Armillaria species are remarkably successful components of many natural forests. A large proportion of tree and shrub species of different strata, particularly in boreal and temperate forests, may be susceptible to *Armillaria* infection. Besides a pathogenic role, members of the genus contribute significantly to decomposition and mineral cycling as well as playing minor roles as mycoparasites and mycotrophic associates with some achlorophyllous plants.

Armillaria in natural forests is endemic, evidence of disease is often obscure, and it may often have minimal effect on host health and growth. However, a continuum of disease effects from non-lethal to lethal involves *Armillaria* species as primary or secondary pathogens. Epidemic disease involving *Armillaria* in either role may result when the balance between pathogenicity and host resistance is altered by stress or disturbance. Many forests may be utilized without aggravating the endemic disease level. In others, disturbance such as logging may lead to a major imbalance between host and pathogen. Inappropriate management in some regions has created a heritage of root disease problems.

Our understanding of the ecology and dynamic behavior of *Armillaria* in natural forests has developed significantly in recent years through better knowledge of species identity, pathogenicity, ecology, and a clear recognition of primary and secondary modes of behavior. In one case at least, it is now feasible to integrate this knowledge through a computer simulation model to better understand root disease dynamics and their response to forest management treatments (see chapter 10). Significant research needs remain. Specifically, future investigations should examine inoculum buildup, quantifying pathogen spatial distributions and dynamics, the relative importance of host, pathogen, and environment on disease expression, and the ecological effects of disease.

CHAPTER 9

Armillaria in Planted Hosts

Ian A. Hood, Derek B. Redfern, and Glen A. Kile

In chapter 8, *Armillaria* was examined in its natural forest environment, modified or not by the activities of man. We now look at the disease in the various artificial habitats created when selected hosts are cultivated either for commercial production or as ornamentals for their aesthetic appeal. The distinction between natural and artificial environments is not always a sharp one, as when seedlings are planted beneath a natural overstory or when new plantations are infiltrated by seed regeneration from nearby stands. Indeed, it is sometimes difficult to decide if a plant growing within (or even outside) its natural range has been planted or naturally seeded. Even so, the planted host generally occurs in a very different setting from its natural counterpart which is adapted to its own particular ecosystem. We might therefore expect *Armillaria* to be frequent and widespread in many species of cultivated plants (Day 1929, Garrett 1956a, Mallet and others 1985).

To demonstrate just how widespread attack by *Armillaria* really is, a broad overview of the disease's geographic distribution begins the chapter. Sixty years of records are used to assess the global importance of *Armillaria* on cultivated plants. An account of the development and impact of *Armillaria* in plantations then follows, and the chapter is concluded by discussing how various management procedures may affect disease development.

Distribution and Importance

The literature on *Armillaria* in planted crops and ornamentals is vast. A large selection of reports has been collated and summarized in tables 9.1 (p. 140) and 9.2 (p. 142), and in fig. 9.1. Together, these present a broad picture of the disease in various host groups throughout the world (for host species lists see, for instance, Spaulding 1961, Raabe 1962a, Hansbrough 1964, Browne 1968). The number of references in these tables indicates the importance of the disease for particular hosts in different countries, with some qualifications: numbers may reflect the regional activity of plant pathologists besides that of the disease, and may be biased toward hosts of greater economic importance. The tables indicate only the presence of the disease, not its severity, which is often expressed only in qualitative terms. Despite these drawbacks, the figure and tables summarize where *Armillaria* occurs in planted hosts throughout the world.

Several trends may be detected in tables 9.1 and 9.2, and fig. 9.1. For example, *Armillaria* is not listed on any major cereals (wheat, rice, maize, oats, barley, rye) or certain other cultivated food crops (peas, beans, groundnut) which are normally cultivated on arable land but rarely, if ever, planted on former forest sites (see later). Records appear weighted in favor of cash-crop plants present in commercial plantations rather than non-commercial hosts used in subsistence cropping or shifting agriculture on cleared forest land. Records for ornamental trees and shrubs also seem under-represented and somewhat fragmentary. Probably many occurrences of the disease in amenity plantings go unreported or are listed only in unpublished records of state agricultural agencies and experiment stations.

Figure 9.1 clearly shows that *Armillaria* is widespread in planted hosts throughout temperate regions and in much of the tropics. Detailed information on disease occurrence in particular regions is normally unavailable but occasionally does exist. For instance, figure 9.2 shows the distribution of 574 *Armillaria*-caused deaths of mainly planted hosts in southern Britain during the past 25 years. Although the frequency of records is biased toward the collection center, and is influenced by the uneven distribution of parks or gardens and former woodland sites, results indicate a widespread occurrence of disease within the survey region. No doubt intensive long-term surveys would present a similar picture where conditions favor the disease elsewhere in the world (Hood 1989).

Lack of detailed information on disease incidence prevents precise comparisons between regions. However,

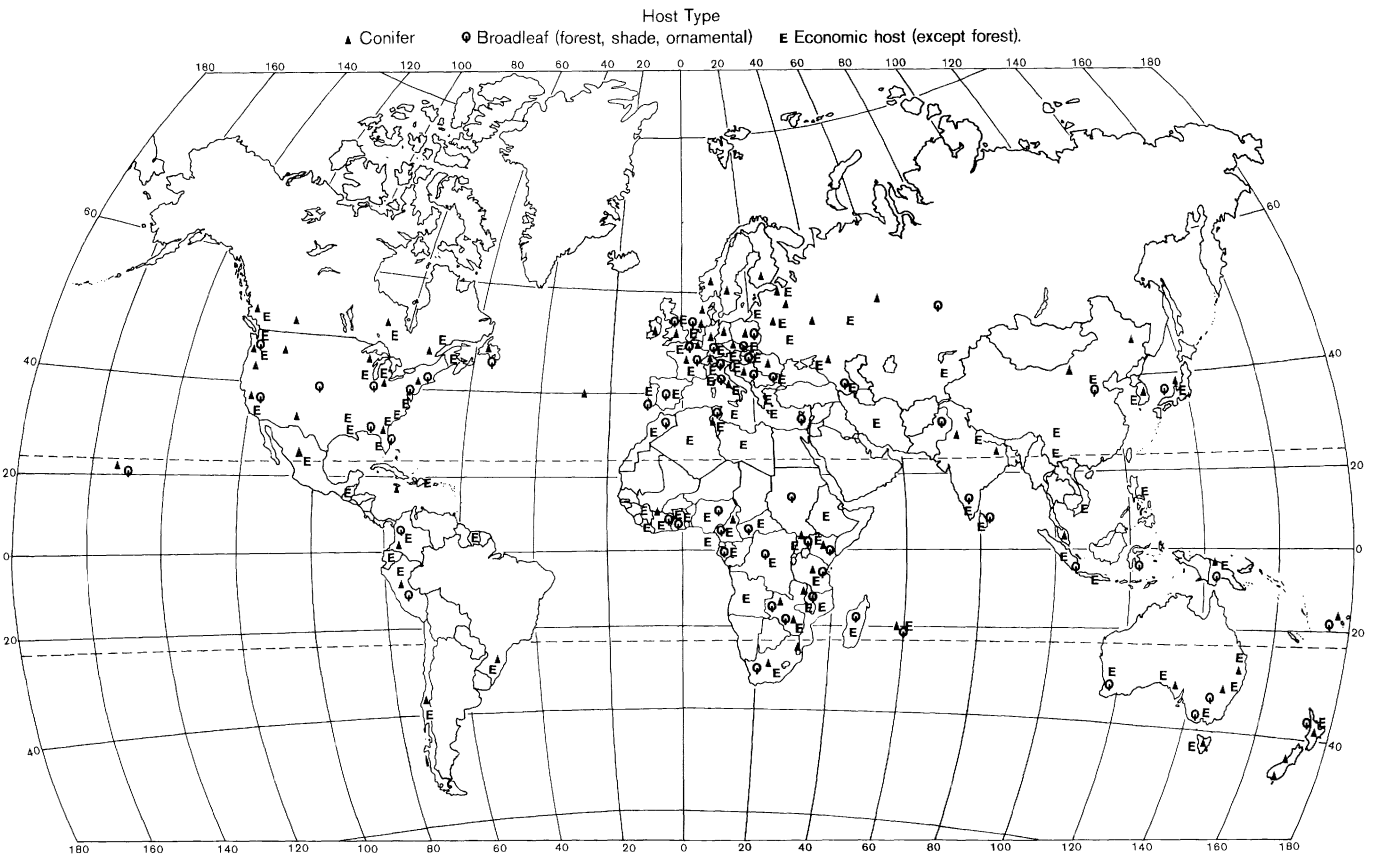


FIGURE 9.1 — Distribution of recorded *Armillaria* attacks in planted hosts, by country (and region).

the literature suggests that the disease is most common in areas with a moist environment and a moderate temperature range (Bliss 1946; Browne 1968; Bunting 1924; Fox 1964; Gibson 1961, 1975, 1979; Ivory 1987; Jie 1982; Jorge 1977; Kile 1980a; Longenecker and others 1975; Mańka 1980; Mohammed and others 1989; Rivera 1940; Rudd-Jones 1950; Rykowski 1980; Sokolov 1971; Tarry 1969). Precipitation and temperature may be the primary factors governing both the altitudinal and latitudinal distributions of *Armillaria*. Thus, in many tropical areas the disease is known only in plantations established at higher elevations where the climate is cooler and wetter (Arentz and Simpson 1989, Barnard and Beveridge 1957, Bernard 1926, Brazilian Inst. Forestry Dev. 1976, Fox 1970, Gill 1963, Ivory 1975, Raabe and Trujillo 1963, Rayner 1959, Satyanarayana and others 1982, van der Goot 1937). This contrasts with certain other tropical root disease fungi which are found mainly in plantations growing at low and mid elevations where the climate is hotter (*Phellinus noxius* (Corner) Cunningham, *Rigidoporus lignosus* (Klotzsh) Imazeki, Fox 1970). In the temperate zones, *Armillaria* attacks plantations established at low and mid altitudes, but not those planted at higher elevations where it is too cold (Johnson 1976, Rahm 1956, Singh and Khan 1979, Twarowski and Twarowska 1959). In the same way, the disease may occur less often at higher latitudes where the climate is colder. *Armillaria* also appears to be absent from certain inland regions with extreme continental climates, as in parts of the Soviet Union (Sokolov 1964).

Europe and the Soviet Union

Europe has a tradition of plantation forestry dating back at least 3 centuries. *Armillaria* is widespread in forest plantations on this continent, and few countries, if any, lack disease records (tables 9.1, 9.2a,b; Day 1929; fig. 9.1). Reports are particularly numerous from France (see also Boullard 1961), Germany, Poland, and the United Kingdom. *Armillaria* is widespread in planted forests in the Soviet Union, (Fedorov and Bobko 1989, Fedorov and Poleschuk 1981, Fedorov and Smoljak 1989, Sokolov 1964) and has also been found on eucalypt species in Spain, Portugal, and Cyprus (Gibson 1975, Ivory 1987). Reports are more numerous for conifer than hardwood stands (tables 9.1, 9.2a).

Much fundamental research has been undertaken in European plantations since Robert Hartig first established the relationship between *Armillaria* and disease

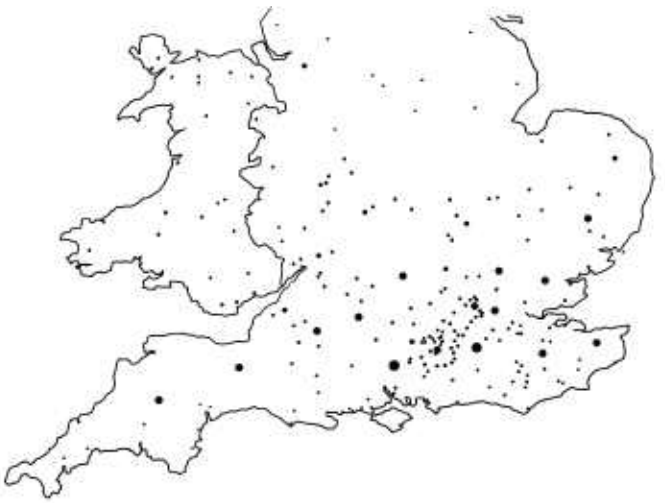


FIGURE 9.2 — Distribution of deaths in planted hosts due to *Armillaria* in southern Britain, 1962-1986. Dot diameter indicates number of records (respectively, over 50, 20-50, 10-20, under 10, in decreasing order of size; data courtesy Pathology Branch Advisory Service, UK Forestry Commission).

in Germany in 1873 (Hartig 1873b, 1874; Nechleba 1915), and this has contributed greatly to our knowledge of the disease's nature and development worldwide. Until recently, most disease records in Europe were attributed to *A. mellea (sensu lato)*, and sometimes *A. tabescens*. We now know (see chapters 1, 2) that attack in European plantations is caused predominantly by *A. mellea (sensu stricto)* and *A. ostoyae* (Korhonen 1978, reviewed Roll-Hansen 1985). This knowledge has encouraged research to define the ecological roles and behavior of the European *Armillaria* species (Guillaumin and Berthelay 1981; Guillaumin and Lung 1985; Guillaumin and others 1984, 1985, 1989a; Holdenrieder 1986; Rishbeth 1982, 1983, 1985a,b, 1987; Siepmann 1985). Recent work shows that at least in western Europe vigorous trees in pure conifer plantations are attacked mainly by *A. ostoyae*. Most infection in planted hardwoods (forest and ornamental trees, table 9.2a) is due to *A. mellea;* in stressed hosts, several less pathogenic species are sometimes responsible (e.g., *A. gallica;* Clancy and Lacey 1986; Davidson and Rishbeth 1988; Durrieu and others 1985; Guyon and others 1985; Intini 1989a,b; Laville and Vogel 1984; Lung-Escarmant and Taris 1985, 1989). More severe attacks in conifers usually occur on former natural hardwood forest sites (often oak) rather than pure conifer stands (Peace 1962, Redfern 1975, Rishbeth 1982, Usčuplić 1980), so that the precise manner in which *A. ostoyae* invades and develops in new conifer plantations requires elucidation (Guillaumin and others 1989a). Guillaumin and Lung (1985) indicated, however, that *A. ostoyae* can grow and produce rhizomorphs quite successfully on hardwood species even though it is a parasite primarily on conifers (see also Davidson and Rishbeth 1988, Gregory 1989, Redfern 1975, Rishbeth 1982).

Although *Armillaria* is considered Europe's most important root disease after *Heterobasidion annosum* (Fr.) Bref., its impact on forest plantations is comparatively minor in this region (Peace 1962). It creates canopy gaps and lessens the returns from early thinnings; but in general, the effects of early mortality are probably over-estimated due to the rather spectacular appearance of the disease in young stands (Pegler and Gibson 1972). Under certain stand or site conditions, damage can become more severe on some hosts. For instance, in Poland *Armillaria* has been responsible for serious losses of Norway spruce and Scots pine (Mańka 1980, 1981; Twarowski and Twarowska 1959; table 9.1).

Armillaria kills ornamental trees and shrubs planted in gardens, parks, and reserves (table 9.2b; Ingelström 1938) and reports are particularly numerous from Great Britain (Boughey 1938, Gibbs and Greig 1990, Greig and Strouts 1983, Peace 1962, Rishbeth 1983, Schilling 1989). The records of mortality shown in fig. 9.2 were predominantly from amenity plantings and were mainly on hosts in the families Pinaceae (31% of records), Rosaceae (21%), Fagaceae (13%), Oleaceae (12%), and Salicaceae (10%).

Food production is far older than plantation forestry, and losses from *Armillaria* must have occurred historically wherever people cultivated plants. Today, the disease commonly attacks many European crops, particularly pip fruit (*Citrus,* Mediterranean countries), pome fruit *(Malus, Pyrus),* stone fruit (*Prunus,* Guillaumin 1977, Guillaumin and Pierson 1983, fig. 9.3), walnuts, and grapes (Guillaumin 1986b, see table 9.2b and Guillaumin 1982, Guillaumin and others 1982, fig. 9.4a). The

FIGURE 9.3 — Mortality gap in almond orchard (on peach rootstock) caused by *A. mellea* with trees dying at the margin; Aigaliers, southern France. (D. Barrett)

A

B

FIGURE 9.4 — A: Mortality gap in grape vineyard, caused by *A. mellea* near Bordeaux, France. (J.-J. Guillaumin). B: Group of trees killed by *Armillaria* sp. in 15-year-old plantation of slash pine; Usa, Tanzania. (M.H. Ivory)

disease has been reported less frequently in cane fruits (Marsh 1952), cork oak, fig, and flower crops (Guillaumin and others 1982), hazelnut, hops, kiwifruit, mulberry, olive, strawberry, various vegetable crops, and in an *Opuntia* cactus crop (grown for its edible fruits). *Armillaria* also attacks various cultivated crops in Lithuania and the Ukraine (Dist. of Plant Dis. 1980), and in Azerbaijan, Armenia, Georgia, Belorussia, Tatar, and eastern Kazakhstan (Sokolov 1964).

Details of *Armillaria* in European plantations have been reviewed by Peace (1962), Pawsey (1973), Schönhar (1977), Rykowski (1980), Guillaumin (1982, 1988), Guillaumin and others (1982), Roll-Hansen (1985), and Phillips (1988). Greig and Strouts (1983) give a popular account of the disease in Britain.

North America

The behavior of *Armillaria* in both managed and unmanaged forests has been extensively researched in North America since early this century (Byler 1984, see chapter 8). In many of these stands, *Armillaria* occurs as an important butt-rot agent and also influences forest successional development by killing seedlings, saplings, and more mature trees, particularly those already weakened by other causes (Byler 1984; Wargo 1980b, 1984b). Most reports of disease in forest plantings are from coniferous stands (table 9.1). Records on hardwoods have come mainly from ornamental or shade trees in the eastern half of the continent (table 9.2a). In conifer plantations in the west, *Armillaria* is one of several important root- and butt-rot disease fungi such as *Phellinus weirii* (Murr.) Gilb. and *H. annosum*, which often occur in the same host or stand. Until recently, all North American records of *Armillaria* were attributed to *A. mellea* (or *A. tabescens*, see below). At least nine biological species of *Armillaria* are now known on the continent, some of which are related to European species (see chapters 1, 2). As in Europe, work is currently underway to define the ecological roles of these species and to identify those which cause disease in plantations and managed stands (Mallet and Hiratsuka 1988, McDonald and Martin 1988, Proffer and others 1987).

Present forests in western North America originated largely by natural seeding following logging of the old growth forests that began during the 19th century. Planting is now carried out to improve stocking levels of desirable species. Early *Armillaria* research in planted forests was done in coastal British Columbia, where trees in young plantations of Douglas-fir are killed by a species now identified as *A. ostoyae* (Bloomberg 1990; Buckland 1953; Hood and Morrison 1984; Johnson and others 1972; Morrison and others 1985a,b; Pielou and Foster 1962). Morrison (1981) reviewed the current understanding of the disease in this province. He considered *Armillaria* to be comparatively unimportant in coastal conifer plantations because mortality is low (1-5%), and ceases after about age 25 years. By contrast, interior British Columbia experiences higher mortality in both plantations and natural, mixed conifer forests (Bloomberg and Morrison 1989; Morrison 1981; Morri-

son and others 1988). In neighboring Alberta and other prairie provinces of Canada, disease impact has not been great, and mortality from *Armillaria* has occurred mainly in naturally regenerated conifers (Blenis and others 1987, Hiratsuka 1987, Mallet 1989). However, deaths also occur in plantations (Emond and Cerezke 1990, Hiratsuka 1987), and Armillaria root disease may become more common as management intensifies (Blenis and others 1987). Preliminary work suggests that the species pathogenic to conifers in Alberta are *A. ostoyae*, a form of *A. cepistipes*, and possibly *A. mellea* (Blenis and others 1987, Mallet 1989, Mallet and Hiratsuka 1988). In Europe, *A. cepistipes* is not considered to be a serious parasite (see chapter 6), and *A. mellea* is most important on hardwood hosts (Davidson and Rishbeth 1988, Guillaumin and others 1989a).

Armillaria root disease (apparently *A. ostoyae*, Filip 1989a, Hadfield and others 1986, Morrison and others 1989) is widespread in western Washington and Oregon, but has the same low impact in conifer plantations as it has in adjacent coastal British Columbia (Filip 1979, Hadfield and others 1986, Johnson 1976). The disease also affects ornamental plants in urban areas in this region (Schmitz 1920). *Armillaria* kills planted ponderosa pine in central Oregon (Adams 1974), but at present it has greater impact in managed natural stands of ponderosa pine and other species in central and eastern Oregon and Washington (Hadfield and others 1986, see chapter 8).

Armillaria is of little consequence in planted and natural coniferous forests in California (table 9.1), but it does attack many ornamental, orchard, and horticultural host species in this State (tables 9.2a,b; see below), suggesting that perhaps different species are involved (Adaskaveg and Ogawa 1990). Although *Armillaria* occurs in conifer plantations in Idaho and New Mexico (Weiss and Rifle 1971, table 9.1), the disease has only been reported from natural forests in Montana, Wyoming, Utah, and Colorado (Wargo and Shaw 1985). *Armillaria* is found across parts of the Great Plains, including North and South Dakota, Nebraska, Kansas, Oklahoma, and the eastern edges of Montana, Wyoming, and Colorado, where it occurs sporadically in over 25 tree species established as windbreaks, Christmas tree, recreational, roadside, and landscape plantings (Fuller and James 1986). The species identification is unknown.

Armillaria is present in Canada's eastern maritime Provinces and has caused disease in planted conifers in Quebec (table 9.1, Magasi 1990). In Newfoundland, it causes serious disease in plantations of native and introduced species of fir, spruce, and pine (Hall and Schooley 1981; Hall and others 1971; Khalil 1977; Singh 1978, 1980a,b, 1981b,c, 1983; Singh and Bhure 1974; Singh and Richardson 1973). Losses may exceed 30% but are usually lower. The disease also affects urban and shade trees in Newfoundland (Singh and Carew 1983). In Ontario, *Armillaria* frequently infects both natural forests (Basham 1958, Hord and Quirque 1956, Whitney and MacDonald 1985) and conifer plantations (Huntley and others 1961; Whitney 1983, 1988b; Whitney and others 1978, 1989a). Losses have not been serious, in some cases because trees were established on abandoned farmland. The species pathogenic to conifers in this province is *A. ostoyae* (Anon. 1989, Whitney and others 1989a). *Armillaria* is also present in Manitoba's forest plantations (C.G. Shaw III, pers. comm.).

Across the southern border, the disease attacks conifer plantations in Minnesota (9% mortality; Livingston and others 1982), throughout Wisconsin (Patton and Riker 1959), and in Michigan (under 5%; Bruhn and others 1989). Several planted stands of red pine in Wisconsin experienced mortality ranging from 10% to 37% at age 10 years after the natural oak overstory was killed by aerial application of herbicide (Pronos 1977; Pronos and Patton 1977, 1978). Although few reports document *Armillaria* in planted hosts in the northeastern USA, the disease is common there and causes losses in forests and Christmas tree plantations (table 9.1, Cook 1961, Longenecker and others 1975, Silverborg and Gilbertson 1962). Ornamental and urban shade trees are also attacked, and *A. mellea* and *A. gallica* both occur on hardwoods in this region (Dunbar and Stephens 1975, Motta and Korhonen 1986). *Armillaria* has little impact in eastern white pine plantations in North Carolina (Leininger and others 1970).

In the southern and southeastern USA (Mississippi, Louisiana, Georgia, Florida) *Armillaria* attacks many different ornamental and shade trees (table 9.2a; Rhoads 1956, Sinclair and others 1987). It and other fungi have reportedly caused up to 25% mortality in plantations of sand pine (Barnard and others 1985, Ross 1970). The pathogen in this region was known as *Clitocybe tabescens* as early as 1930 (table 9.2a), and the disease is still attributed to *A. tabescens* in current reports (Sinclair and others 1987) although further examination is needed to establish its identity (Guillaumin and others 1989a).

For reviews of *Armillaria* in North American forests, including plantations, see Boyce (1938), Singh (1980b), Wargo and Shaw (1985), and Sinclair and others (1987). Hepting (1971) supplied information for individual hosts, and a popular account of the disease was recently prepared by Williams and others (1989).

Armillaria severely affects orchard and horticultural crops in North America, particularly in California, Florida, and the Pacific Northwest (Washington, Ore-

gon, and British Columbia). In California, it is considered one of the most serious diseases of stone fruits (Wilson and Ogawa 1979), and it also infects citrus, walnuts, and grapes (table 9.2b). The disease has affected these crops since the turn of the century on sites previously forested with native oaks, giving rise to the name "oak root fungus" (Gardner and Raabe 1963, Hewitt 1936). *Armillaria* infects California's pome fruits (table 9.2b), but it is apparently unimportant in apple and pear orchards (Wilson and Ogawa 1979). The disease has also been recorded on California's avocado, blackberry, fig, kiwifruit, loquat, persimmon, and strawberry crops (table 9.2b). Wilson and Ogawa (1979) included olive, chestnut, hazelnut, commom pistachio, and common pomegranate as additional hosts in the State.

In Washington and Oregon, hosts include currants, gooseberries, hazelnuts, hops, pome fruits, raspberries, stone fruits, strawberries, and walnuts (table 9.2b, Childs and Zeller 1929, Lawrence 1910, Piper and Fletcher 1903). *Armillaria* occurs in British Columbia's pome and stone fruits, and occasionally its raspberries and potatoes (table 9.2b). In Florida, *Armillaria* (cited as *"A. tabescens"*) has been reported on citrus (Rhoads 1948), and stone fruit trees have been attacked in several southeastern States (table 9.2b; Weaver 1974). Other host crops in Florida have included banana, grape (also in Missouri), guava, litchi, pome fruits (also in Louisiana), and tung (also in Louisiana). Pecan trees have been attacked in Georgia, and stone fruit trees in Wisconsin, Illinois, Michigan, Ontario, and Quebec. The disease is recorded on fruit trees in the Eastern States of North Carolina and Maryland (Cooley 1943).

Little work has been done to identify which species of *Armillaria* attack horticultural and fruit crops in North America. In Michigan, Proffer and others (1987) found *A. ostoyae, A. mellea,* and a species since described as *A. calvescens* (Bérubé and Dessureault 1989) attacking sour cherry trees planted on susceptible root stocks (see also Adaskaveg and Ogawa 1990).

Central and South America

Records of disease on planted hosts are much less frequent from Central and South America, but they suggest that *Armillaria* has a wide, if sporadic, distribution in both the tropic and temperate zones throughout the region (table 9.1, 9.2a,b).

In warm-temperate, northern Mexico, *A. ostoyae* has caused root disease in radiata and Arizona pine plantations near Chihuahua (Hawksworth 1987, Shaw 1989a).

Armillaria occurs in tropical America and has been recorded on conifers in Jamaica and Peru, mainly on pines such as slash pine (table 9.1). According to Ivory (1987), pines have been attacked in Cuba, Honduras, Surinam, and Ecuador. C.A. Garzon B. (pers. comm.) has observed mortality caused by *Armillaria* among planted eucalypts at high elevation near Popaýan in western Colombia. The disease was reported in Colombia between 1975 and 1982 in slash pine, Mexican weeping pine, Mexican cypress, teak, and eucalypts (C. Alvarado, J.J. Castaño, M. Gutierrez, E.R. Ordoñez, M.; per C.A. Garzon B.), but it is not considered to be important in that country. One account documents *Armillaria* on hardwoods in Peru (table 9.2a), but it is not recorded in the extensive eucalypt, Gmelina, and pine plantations that have replaced natural rainforest in the Amazon Basin of Brazil, suggesting that the disease currently has little, if any, significance in the hot, lowland climate of this region (Brazilian Inst. Forestry Dev. 1976, Rankin 1985).

Few records implicate the disease among cultivated crops in tropical America. *Armillaria* has been reported on cacao in Mexico, Colombia, and possibly Brazil; and it is listed on avocado in Ecuador (table 9.2b; Wood and Lass 1985). Locally, *Armillaria* has infected Cinchona plantations in Peru (table 9.2b) and economic food crops in the Dominican Republic, Guatemala, and Surinam (Dist. of Plant Dis. 1980). However, it is not notable as a problem in cacao in Ecuador (J. Hedger, pers. comm.), and it was not detected in Trinidad and Venezuela (Dennis 1950, 1970). Nothing is known of the species responsible for disease in tropical America.

In temperate South America, *Armillaria* has been reported from Chile and southern Brazil on introduced conifers, especially pines (table 9.1). The disease has killed groups of radiata pine less than 6 years old in Chile, but 1% or less of the forest is affected (H. Peredo L., pers. comm.). Reis (1974) noted that *Armillaria* had been recognized for a number of years in pine plantations in Chile (radiata pine) and Brazil (slash, loblolly, and Mexican weeping pine; May 1962a,b, 1964; Brazilian Inst. Forestry Dev. 1976). Gibson (1973) observed minor damage in slash pine plantations up to 12 years old near São Paulo. In its warmer, northerly range in southern Brazil, the disease is confined to higher elevations (Brazilian Inst. Forestry Dev. 1976). Mortality varies between 1% and, rarely, 25-30%, and ceases after age 5 years (Brazilian Inst. Forestry Dev. 1976, Ferreira 1989, Hodges 1971). Among cultivated plants, *Armillaria* has been reported on cassava and pome fruits in Chile and grapes in southern Brazil (table 9.2b; May 1962a). No published records of the disease so far exist for plantations in Argentina (J.E. Wright, pers. comm.).

Many *Armillaria* species have been described from temperate South America, including *A. limonea* and *A. novae-zelandiae* which are pathogenic in Australasia

(Horak 1979; Kile and Watling 1983; Shaw and Calderon 1977; Singer 1953, 1969; Spegazzini 1922), but the species causing disease in pine plantations and horticultural crops have not been identified. However, the Northern Hemisphere species are very likely absent or of only minor importance in the south-temperate regions of the world.

Africa

Africa has provided many records of *Armillaria* in various planted hosts from numerous localities (tables 9.1, 9.2a,b; Ivory 1988). These are scattered from the north-temperate zone (Morocco, Algeria, Tunisia, Libya) across the tropics to temperate southern Africa (fig. 9.1). The impact and intensity of disease varies with location and host. A number of reviews, surveys, and literature collations have been published (Browne 1968; Fox 1970; Gibson 1967, 1975, 1979; Ivory 1987; Mohammed and others 1989). According to Mohammed and others (1989; *cf* Mwangi and others 1989), the two most common species on planted hosts are *A. heimii*, which was collected mainly above 2,000 m in eastern, central, and southern Africa, and a species culturally close to *A. mellea* which was found at lower altitudes on both sides of the continent (Pegler 1977). Both occurred on hardwood and conifer plantation species and on horticultural crops. These distributions are interesting because *Armillaria* tends to infect plantations established above 1,000 m in eastern Africa where the climate is cooler and wetter. In central and west Africa (Ghana, Nigeria, Zaire, Uganda), the disease occurs in crops at both high and low altitudes (Blaha 1978, Fox 1970). Further surveys are needed, however, before the various altitudinal occurrences of disease on each side of the continent can be considered species-related.

Plantations of fast-growing hardwood and softwood trees have been established in many African countries in order to replenish natural timber resources that are gradually becoming exhausted (Gibson 1967; Ofosu-Asiedu 1980, 1988; Wingfield 1987). Losses from *Armillaria* have been recorded in species of *Araucaria, Pinus, Widdringtonia, Acacia, Albizzia, Cassia, Cedrela, Cupressus, Eucalyptus, Gmelina, Grevillea, Khaya, Tectona, Terminalia, Toona,* and *Vitis* (Gibson 1967, 1975; L.M. Mwangi, pers. comm.; fig. 9.4b). Other reports supplementing those listed in table 9.1 and 9.2a are supplied by Gibson (1964, 1967, 1975); Scharif (1964); Browne (1968); Saccas (1975); Bakshi (1976); Ofosu-Asiedu (1980); Nandris and others (1984); Piearce (1976, 1984); Nicole and Mallet (1985); and Chipompha (1987). These document additional location records of the disease in planted forest species in Cameroon, Central African Republic, Gabon, Ivory Coast, Morocco, Nigeria, Sudan, Swaziland, and Zambia (fig. 9.1). In West African countries, incidence of disease is normally infrequent and occurrence is confined to localized infection centers, but Gibson (1967) reported up to 60% loss of *Albizzia falcata* in Gabon. In North Africa the disease has been reported from Tunisia (eucalypts, occasional deaths, Gibson 1967) and Morocco (teak, up to 20% losses on some sites from *Armillaria* and *Rigidoporus lignosus* (Kl.) Imaz, Gibson 1967).

Armillaria has been investigated intensively in forest plantations in Kenya, Malawi, and Zimbabwe (Chipompha 1987; Gibson 1957a,b, 1960, 1961; Gibson and Corbett 1964; Gibson and Goodchild 1960; Masuka 1989; Olembo 1972; Olembo and others 1971; Swift 1968, 1970, 1972). In these countries, attack occurs on cool, moist, higher sites formerly occupied by natural rain forest rich in hardwood species (Leach 1939) or on old hardwood plantation sites (Gibson 1979). Overall, the impact of the disease is minimal, but it can be locally severe (over 30% mortality) in younger stands of susceptible species such as teak, slash pine, and mlanji cedar. Normally of little or only local significance, *Armillaria* infects plantations of pine, teak, and occasionally other species in Uganda, Tanzania, Zambia, Swaziland, and South Africa (Gibson 1964, 1967, 1975; Kotzé 1935; Lückhoff 1964; Lundquist 1986, 1987; Lundquist and Baxter 1985; Piearce 1984; Wingfield 1987; Wingfield and Knox-Davies 1980; Wingfield and others 1989).

The horticultural crops most commonly attacked in tropical Africa (table 9.2b) have been cacao (countries bordering the Gulf of Guinea, Central African Republic, Zaire, Uganda and Madagascar; Saccas 1975), coffee (widespread; Guinea to Mauritius, Ethiopia to Zimbabwe; Blaha 1978), tea (central and east Africa from Zaire to Mauritius, Kenya to Zimbabwe; Fassi 1959; Leach 1937, 1939), and rubber (central Africa from Liberia to Uganda; Fox 1964, 1970; Mallet and others 1985; Wastie 1986; D. Nandris, pers. comm.). Brief reviews of the disease have been published for cacao (Thorold 1975, Wood and Lass 1985), and for tea and coffee (Haarer 1963, Saccas 1975, Wallace 1935). In tea and coffee, the disease is known as "collar crack disease."

Armillaria has been reported less frequently on the following cultivated hosts in Kenya, Uganda, Tanzania, Malawi, Zimbabwe (table 9.2b; Saccas 1975, Wiehe 1952): banana, cassava, Cinchona, citrus, fig, guava, geranium/pelargonium, granadilla, loquat, macadamia nut, mango, olive, papaya, pome fruit, stone fruit, sugar cane, and tung. Additional hosts in Zimbabwe are avocado, cotton, grapes, pecan, and strawberry (F.A. Chanakira-Nyahwa, pers. comm.). Records elsewhere in tropical Africa (table 9.2b; Saccas 1975, Turner 1970, Wardlaw 1965) include avocado (Ghana), banana (Ghana), cassava (Ivory Coast, Central African Republic,

Zaire), *Cinchona* (Guinea, Zaire), coconut (Ghana), *Cola acuminata* (Ghana), cotton (Zaire), *Hydnocarpus anthelmintica* (Zaire), lime (Ghana), mango (Ghana), mulberry (Central African Republic), oil palm (Ghana, Zaire), and pome fruit (Zaire). Wardlaw (1972) reviewed the disease in banana. Further records of *Armillaria* in cultivated plants are listed from Angola, and possibly Egypt and Sierra Leone (Dist. of Plant Dis. 1980). In temperate Africa, *Armillaria* has been recorded on fig and citrus in the north (Algeria, Libya, Morocco, Tunisia), and from banana, citrus, stone, and pome fruit trees in South Africa (Wingfield 1987).

A variety of tropical ornamental trees and shrubs are subject to attack from *Armillaria* in Zimbabwe (F.A. Chanakira-Nyahwa, pers. comm.), and the same is no doubt true of other countries in Africa.

Asia and the Pacific

Records of *Armillaria* in planted hosts are widespread, but sporadic, from the Middle East to the Pacific Ocean, even though parts of this region (China, Japan) have a centuries-old reforestation tradition (Izumi 1988, Winters 1974). In north temperate Asia, the disease has been reported in economic crops from Iran and apparently Iraq (Dist. of Plant Dis. 1980, Bakshi 1967) and in conifer plantations or hardwood trees from Pakistan, India, China, Korea, and Japan (table 9.1; Bakshi 1967). A record from cypress in Lebanon may refer to a plantation (Scharif 1964), and in northern Pakistan, infection has been observed on persimmon (Zakaullah and others 1987). *Armillaria* occurs in the western Himalaya Ranges in northern India (Bakshi 1976, 1977) but caused less than 3% mortality in young plantations of deodar cedar and pindrow fir (Singh and Khan 1979). Further east, *Armillaria* has been found in exotic pine and Japanese redcedar plantations in northern West Bengal (Bakshi 1976, Singh and Khan 1982).

Armillaria is widespread in China (Zhang and Huang 1990). Jie (1982) described extensive attack in Heilong-Jiang province in the northeast affecting plantations of Korean pine and larch. She reported *Armillaria* in the interior provinces of Gansu, Sichuan, and Yunnan, on both broadleaf and conifer hosts. *Armillaria* infects planted Korean pine trees but is not considered serious in Inner Mongolia province (Yang Li, pers. comm.). In Hebei province the disease is attributed to *A. tabescens* and is recorded in fruit, ornamental, and woodlot trees (apple, pear, peach, almond, white mulberry, locust, poplar, willow, elm, ailanthus, and jujube; Chang and others 1982). *Armillaria* is a serious problem on citrus in Sichuan province, and on tea and cocoa in Yunnan province in the south (Beijing Forestry University 1983).

Armillaria occurs throughout Japan and kills trees in young plantations of Japanese larch (Bakshi 1967; Imazeki 1964; Kawada and others 1962; Ono 1965, 1970) and hinoki (Muramoto 1987, 1988; Terashita and others 1983). Mortality rates for hinoki in Kagoshima Prefecture (Kyushu) are mostly under 10% (M. Muramoto, pers. comm.). *Armillaria* has been reported on pine in Japan (Bakshi 1976; Kitijima 1934). It also affected a cherry orchard near Osaka (Aoshima and Hayashi 1981). According to Guillaumin and others (1989a), *A. mellea* is one of the species commonly present in southern Japan where it occurs mainly on non-conifers although three isolates were obtained from hinoki. A Sakhalin spruce collection made from northern Japan was identified as *A. ostoyae*. This and other species have been reported from northern India (Chandra and Watling 1981, Watling and Gregory 1980), but generally more work is needed to clarify our knowledge of the pathogenic species inhabiting plantations in temperate Asia (see chapters 1 and 2).

Armillaria also infects coniferous and hardwood hosts in Korea (Bakshi 1967, Imazeki 1964, Lee and others 1987). The disease occurs in plantations of Korean pine (Sung and others 1989) and also mulberry plantations and orchards (Office of Forestry 1969).

In tropical Asia, reports of disease are few, and while this may reflect less disease research in some countries, *Armillaria* causes little or no impact in lowland areas over much of the region. It is rare in tropical India (Ivory 1988), but *Armillaria* has affected green wattle in the south (Nilgiris hills) and *Acacia* and *Albizzia* in Sri Lanka (table 9.2a; Gibson 1975). Petch (1910, 1928) studied the disease in Sri Lankan tea plantations and found that stumps of interplanted *Acacia* species fostered disease spread. Tropical crops such as cacao, coffee, and tea are commonly cultivated beneath a canopy of quick-growing hardwood shade trees such as *Acacia, Casuarina, Gliricidia,* and *Leucaena* (Wood and Lass 1985). Generally, however, *Armillaria* has little impact in Sri Lanka and is not normally seen in horticultural crops there (A. de S. Liyanage, pers. comm.). *Armillaria* was reported on tea in southern India several times since 1960 (table 9.2b), but a record on tung has been disputed (table 9.2b). The pathogenic species in tropical India and Sri Lanka has been described as *A. fuscipes* which may be identical to or closely related to the African *A. heimii* (Chandra and Watling 1981, Kile and Watling 1988, Pegler 1986).

Armillaria has been recorded in Vietnamese (Dist. of Plant Dis. 1980) and Philippine crops (Saccas 1975, Mallet and others 1985, Dist. of Plant Dis. 1980). Reports concerning conifer hosts in the region are few, but *Armillaria* has caused low levels of disease in plant-

ings of Khasi pine and Bahaman pine in the Cameron Highlands of Peninsular Malaysia (Barnard and Beveridge 1957; Ivory 1972, 1975; M.H. Ivory, pers. comm.). In Indonesia, *Armillaria* (sometimes as *A. fuscipes*) was reported several times in the 1920's and 1930's in *Cinchona*, tea, coffee, and citrus in Sumatra and Java. These records were frequently at higher elevations. *Armillaria* has been found in planted hardwood tree species such as *Albizzia*, *Leucaena*, green wattle, and teak in Java and Sulawesi (table 9.2a,b; Gibson 1975, Hadi 1977, Imazeki 1964), but the disease is not common in Indonesia today (S. Hadi, pers. comm.).

Records from Malaysia and Indonesia appear restricted to higher elevations, suggesting that *Armillaria* naturally inhabits the cooler, more temperate, montane forest types rather than lowland, tropical rainforests in this region (Fox 1970). In Peninsular Malaysia, *Armillaria* is absent from lowland plantations of rubber, oil palm, Caribbean pine, *Acacia mangium*, *Gmelina arborea*, and *Paraserianthes falcataria* (R.A. Fox; K.H. Chee; A.M. Tan; Lee S.S.; Norani Ahmad; Maziah Zakaria, pers. comms.).

In Papua New Guinea, too, *Armillaria* occurs only at mid or higher elevations where it causes root rot and mortality in planted pines and eucalypts (F. Arentz, pers. comm., Arentz and Simpson 1989, Shaw 1984). J.A. Simpson (pers. comm.) observed the disease in eucalypt plantations (swamp mahogany and southern blue gum) established mainly on certain sites cleared of natural southern-beech forest. Mexican weeping pine was also killed by *Armillaria* at Marafunga. Other eucalypt species (e.g., flooded and New Guinea gums) showed no evidence of disease on most sites, and infections in pines (Khasi, Mexican weeping, and Honduran at Lapegu and Kainantu) caused no economic loss. *Armillaria* has been recorded in Papua New Guinea on several cultivated crops such as coffee (Shaw 1984; table 9.2b), but is of little economic significance on these hosts. *Armillaria* collections made in Papua New Guinea have been identified as *A. mellea*, *A. fellea*, and *A. heimii* (Shaw 1984), and according to J.A. Simpson (pers. comm.), southern blue gum was attacked by *A. novae-zelandiae*, and swamp mahogany by *A. fellea*.

Few records document the disease in the tropical Pacific (Dingley and others 1981). *Armillaria* attacks Fiji's high- and low-elevation plantings of mahogany and slash pine on former rain forest sites (Singh 1978, Singh and Bola 1981; pers. obs.). It also occurs in the Solomon Islands (Corner, in McKenzie and Jackson 1986) but has not been reported in plantations. In Hawaii, *Armillaria* causes disease in young pine plantations over 1000 m elevation, and it also occurs in planted hardwood hosts (Laemmlen and Bega 1974, Raabe and Trujillo 1963). The species responsible for the disease in these islands is unknown.

Australasia

Armillaria is of little consequence on planted hosts in tropical Australia but is widespread in the less arid parts of the temperate and subtropical regions. Even so, reports of disease have been infrequent in planted forests (table 9.1, 9.2a; Kile 1980a). Locally severe outbreaks have occurred in radiata pine in South Australia (table 9.1) and Tasmania (Kile 1980a), mountain ash in Victoria (Podger and others 1978), and slash, Honduran, and radiata pines in southern Queensland (Bolland and Brown 1981). Mortality declines in pines after about 5 years, and infection centers are small. All planted conifers (pines, Douglas-fir, and Queensland kauri) and hardwoods (eucalypts) appear susceptible to some degree (fig. 9.5), but the overall impact is minor. Kile (1980a) suggested that this may be because much of the newly planted land was converted from farming to forestry or from a natural cover of drier eucalypt forests in which *Armillaria* has a limited distribution. It is

FIGURE 9.5 — Mortality gaps in plantation of alpine ash, caused by *A. luteobubalina*. Mt. Disappointment, Victoria, Australia. (G.A. Kile)

also possible that Australian *Armillaria* species have a low pathogenicity toward many introduced tree species.

Armillaria has been more frequently reported on ornamentals and garden plants, mainly in New South Wales (table 9.2a), Victoria (Smith and Kile 1981), and Western Australia (Kile and others 1983). The disease is considered serious in Melbourne and Sydney, and losses have been recorded in the botanical gardens of Perth, Adelaide, Canberra, and Sydney (Kile unpubl., Kile and Watling 1988, Smith and Kile 1981). Attacks have also occurred in orchards and horticultural crops (table 9.2b), particularly in citrus, pome, and stone fruit orchards in most states (Doepel 1962, Kable 1974, Nicholls 1915). Other listings (table 9.2b) include attacks on bananas (New South Wales), raspberries and loganberries (Tasmania), grapes (Western Australia), hops (Tasmania), mulberry (Western Australia), passionfruit (Western Australia), and vegetables in most states (Doepel 1962, Lea 1909). The species responsible for the disease in urban gardens, fruit orchards, vineyards, horticultural crops, and ornamental trees in Australia is *A. luteobubalina* (Kile and Watling 1988, Kile and others 1983, Smith and Kile 1981). *Armillaria luteobubalina* also caused mortality in planted mountain ash in Victoria (Podger and others 1978). In southern Queensland's forest plantations, *A. novae-zelandiae* and *A. fumosa* have caused minor disease in radiata pine, and *A. pallidula* has been associated with slash and Caribbean pines (Kile and Watling 1988). Earlier records ascribed to *A. elegans* in Australia refer to *A. luteobubalina* (Kile and Watling 1988).

Armillaria occurs throughout New Zealand, and frequently kills woody hosts in parks and gardens. Reports on horticultural crops are less numerous (table 9.2b; Dingley 1969, Pennycook 1989), and according to Atkinson (1971), *Armillaria* is not important as a cause of disease in fruit orchards. *Armillaria* has infected stone and pome fruit trees in the Auckland district and on the South Island, but the impact has been comparatively minor even though individual growers have occasionally sustained heavy losses. *Armillaria* is rare in citrus fruit (Atkinson 1971), but recently, the disease has become serious in many orchards of kiwifruit on the North Island (Horner 1985, 1987, 1988, 1990a,b). Attacks originate from the stumps of felled shelterbelt trees which act as inoculum sources (fig. 9.6). The species causing disease in kiwifruit orchards is *A. novae-zelandiae* (I.J. Horner, pers. comm.).

Both *A. novae-zelandiae* and *A. limonea* are responsible for root disease in radiata pine planted throughout New Zealand where indigenous podocarp-hardwood or southern-beech forests have been cleared (fig. 9.7). Losses are spectacular in the first 5 years with up to

FIGURE 9.6 — Mortality gaps in kiwifruit orchard caused by *A. novae-zelandiae*. Te Puke, New Zealand. Gaps follow lines of stumps of willow shelterbelt trees felled 5 years earlier. (I.J. Horner)

FIGURE 9.7 — Mortality gaps where young trees have been killed by *A. novae-zelandiae* and *A. limonea* in a plantation of radiata pine on a site cleared of indigenous podocarp-hardwood forest, but without stump removal (see fig. 11.1). Tuararangaia Forest, Raungaehe Range, Bay of Plenty, New Zealand. (J. Barran)

30% mortality, but may be more severe later in the rotation in the form of growth reduction and uprooting of final crop trees (MacKenzie 1987). *Armillaria* also occurs in second-rotation forests; however, its significance in these stands is unknown, but perhaps is greater than previously thought (MacKenzie and Self 1988, van der Pas 1981a).

Since the early studies of Birch (1937) and Gilmour (1954, 1966b), much has been learned about disease development in forest plantations under New Zealand conditions (Benjamin and Newhook 1984a,b; Hood and

Sandberg 1987, 1989; MacKenzie and Shaw 1977; Roth and others 1979; Shaw and Calderon 1977; Shaw and Toes 1977; Shaw and others 1976b, 1980, 1981; van der Pas 1981a,b; van der Pas and Hood 1984). This information was recently reviewed (Hood 1989), and popular accounts of the disease are available (Shaw 1976, van der Pas and others 1983).

Disease Development and Impact

Although *Armillaria* occurs in many hosts and places, the same principles govern the behavior of the disease in most plantations throughout the world. This section examines the distinctive features of plantations that influence disease development and describes the effect of infection on crop production. For this discussion, a plantation is defined as a stand or crop created by sowing seed or by planting. Coppice stands derived from adventitious shoots or suckers and forests regenerated beneath seed trees after logging are excluded. Some factors governing disease development in amenity plantings have been considered by Miller (1940), Rhoads (1956), and Rishbeth (1983).

The Significance of Plantations

Plantations differ to a greater or lesser extent from natural forest in several respects. They are often even-aged monocultures in which plants are regularly spaced at an appropriate stocking density. Various forms of selection, including clonal propagation, may give rise to planted stock with a reduced genetic base. These features are intended to facilitate crop management and ensure high product yield. However, some aspects of plantations may encourage disease.

Inoculum Potential

Attack by *Armillaria* invariably involves inoculum in the soil consisting of woody material colonized by the fungus (see chapter 4). In natural, unmanaged forests, such a food base normally becomes available sporadically as trees uproot or are killed by *Armillaria* or other agents. In plantations, by contrast, colonized stumps or debris left after harvesting a previous stand, generally by clearfelling, are particularly abundant when the crop is established and the young plants are most vulnerable.

Induced Host Stress

Root systems of naturally established seedlings growing under favorable soil conditions are normally well formed whereas those of planted seedlings are often deformed or injured. Seedlings weakened in this way are more likely to die from *Armillaria* infection than are vigorous, unstressed seedlings (see chapter 7). Singh and Richardson (1973) observed a higher incidence of mortality among bare-rooted stock than among container-grown seedlings after planting. Kessler and Möser (1974) found that plants established from seed survived *Armillaria* attack during drought stress better than planted trees (see also Buckland 1953, Thies and Russell 1984, Weissen 1981, Whitney and Timmer 1983).

Choice of Species

Planted hosts are often established outside their natural range, and may therefore be exposed to species and strains of *Armillaria* which they would not naturally encounter. Under these circumstances, introduced plants in plantations and gardens may conceivably be more prone to attack than hosts indigenous to the region although evidence to support this hypothesis is meager. Exotic spruce and firs in a Newfoundland plantation proved more susceptible to *Armillaria* than indigenous species of the same genera (Singh and Richardson 1973). In Californian walnut stands, the introduced Persian walnut is susceptible to the local species of *Armillaria*, and is therefore grafted onto rootstocks of the resistant, indigenous northern Californian walnut (Wilson and Ogawa 1979). Either exotic hosts may be inherently susceptible, or suceptibility may be induced by environmental features to which they are not adapted.

Monocultures

Deaths from *Armillaria* will be more numerous where greater numbers of susceptible plants occupy an infected site. Establishing even-aged, uniformly stocked plantations of susceptible species creates an extreme situation conducive to disease expression that may not arise in floristically and structurally diverse natural forests. Moreover, the uniform, close spacing of many monocultures may facilitate disease spread between susceptible plants. Fedorov and Poleschuk (1981) attributed greater disease impact from *Armillaria* and *H. annosum* in the Soviet Union to large-scale planting of single-species forests (*cf* Garrett 1956a). The general principles of disease risk in monoculture plantations have been discussed elsewhere (e.g., Gibson and Jones 1977, Peace 1957).

Disease Dynamics

Disease Establishment

Outbreaks of disease typically occur in crops or plantations that replace natural forests or earlier plantings (fig. 9.7). Inoculum consists of residual infection derived from the original forest or previous crop. This builds up on stumps and root debris (fig. 9.8; see chap-

FIGURE 9.8 — Stump of recently felled tawa tree with root system colonized by *A. limonea* on site cleared of indigenous podocarp-hardwood forest prior to burning and planting in radiata pine (see fig. 9.7). Near Rotorua, New Zealand. (I.A. Hood)

ter 4), from which it spreads to the new plants. *Armillaria* is rare in plantations established on non-forested areas such as grasslands, or arable land that has been cultivated for many decades (Gibson 1957a,b; Huntly and others 1961; Kile 1980a; Liese 1939; Rhoads 1925; Singh 1981c), although disease occasionally occurs on these sites (Fedorov and Poleschuk 1981; Gilmour 1954; Rishbeth 1978b, 1988; van der Pas 1981a). Even under these circumstances, some form of woody material such as a thinning stump is needed to establish the primary inoculum (Swift 1972). Less commonly, primary inoculum may consist of colonized wood material transported in floodwaters (Dadant 1963b, Hewitt 1936, Magnani 1978) or during land contouring prior to planting (Horner 1987). Inoculum may be introduced on wooden stakes, posts, or infected nursery stock (Kable 1974). The fungus can also invade plantations from infected trees or shrubs established for shelter or shade, or as a source of green mulching material, in both tropical (Colonial Research Pesticides Unit 1959; Dadant 1960, 1963b; Fassi 1959; Gadd 1940; Gibson and Goodchild 1961; Leach 1936; Milimo 1989; Petch 1922, 1928; Rishbeth 1980) and temperate crops (Beaumont 1954; Chapot 1964; Horner 1987, 1988; Smith 1971).

Although the role of basidiospores has been disputed (Fox 1970, Kable 1974, Shaw 1981a, Swift 1972), current evidence indicates that the fungus may enter plantations in this form (Rishbeth 1964, 1970, 1987). Airborne basidiospores appear unable to infect living trees directly, with or without wounds (Roll-Hansen and Roll-Hansen 1981, Rykowski 1980), but they can colonize freshly cut wood during the fruiting season (Hood and Sandberg 1987, Molin and Rennerfelt 1959). Basidiospores, which may be less ephemeral than previously assumed (Shaw 1981a), may invade stumps or other debris from which infection then spreads to adjacent, living trees (Fedorov and others 1985, Garrett 1956a, Horner 1988, Petch, in Rishbeth 1955, Rishbeth 1985b).

In theory, new disease centers could be created whenever suitable woody substrates become available during a rotation. When a plantation is established, incoming spores may supplement existing inoculum derived from the previous clearfelled forest, especially if this is substantially reduced during burning of the logging debris and slash prior to planting (Hood and Sandberg 1989, Sokolov 1964). Hot fires may kill still-living stumps, rendering them more susceptible to subsequent colonization. Thinning stumps (Fedorov and others 1985; Peace 1951, 1962; Schönhar 1973) or stumps of shelter trees (Horner 1987) readily harbor the fungus and may act as sources of basidiospore-derived primary inoculum later in the rotation. In Britain, indirect but convincing evidence for basidiospore infection has been demonstrated by the occurrence of small, single-genotype clusters of *Armillaria* infection centered on thinning stumps in first rotation stands planted on former arable or heathland (Rishbeth 1978b, 1985b, 1988). In New Zealand, Horner (1988, pers comm.) has shown that infection centers in kiwifruit orchards are initiated by spores that colonize chemically killed stumps in felled willow shelterbelts (fig. 9.6).

Armillaria infects the new crop when roots of established plants encounter the primary inoculum, either through direct contact or by rhizomorphs. Infection is governed by host susceptibility, pathogenicity of the species or strain of *Armillaria*, and the frequency of root or rhizomorph contacts (see chapters 4 and 6). Rhizomorphs grow out from the inoculum source and are found mainly in the top 20 cm or so of soil (Redfern 1973; Rykowski 1981c; Singh 1978, 1981b), although in some light soils they may live in colonized stump roots more than 2 m beneath the surface (Horner 1987). They can extend laterally up to 5 m from the inoculum source (Peace 1962), but the distance over which they are able to infect host plants is probably much less (see

chapters 4 and 6). In some situations, they may serve as a bridge between roots nearly, or actually, in contact (Kable 1974).

The extent of rhizomorph development depends primarily on the species of *Armillaria* (Guillaumin and others 1984, 1989a; Rishbeth 1985b); but the soil also has an effect (see chapters 4 and 6). Rhizomorphs are rare or infrequent in certain plantations in Southeastern United States (Rishbeth 1978a, Sinclair and others 1987), low and mid-elevation parts of Africa (Anon. 1953, Bottomley 1937, Boughey and others 1964, Fassi 1959, Fox 1970, Kotzé 1935, Olembo 1972, Swift 1968, Wiehe 1952), northern India (Singh and Khan 1979), eastern China (Chang and others 1982), Papua New Guinea (J.A. Simpson, pers. comm.), Fiji (S. Singh 1978), and Australia (Pearce and Malajczuk 1990a, Podger and others 1978).

Disease Distribution Pattern

In young plantations, infected plants typically occur in groups centered on the primary inoculum (James and others 1982, Jie 1982, Peace 1962, Podger and others 1978, Swift 1972, van der Pas 1981b, Zondag and Gilmour 1963). These groups are referred to as infection centers or disease foci. The number of dead trees in a focus is often small (Bolland and Brown 1981, Gibson 1973, Greig and Strouts 1983, Podger and others 1978, Whitney 1983). In these circumstances, the impact of mortality is probably comparatively minor since the limited land area temporarily lost to production is soon reclaimed as surviving tree root systems grow and reoccupy the site (Johnson and others 1972). However, disease centers may be larger and more significant.

The shape, size, and distribution of disease foci are governed both by the spatial occurrence of *Armillaria* in the previous stand or forest and by the distribution pattern of the residual stumps. In western North America, pathogenic *Armillaria* exists in large (often over 400 m across), centuries-old colonies (fig 8.6) in natural ponderosa pine forests (Anderson and others 1979, Shaw and Roth 1976). The same may be true in old-growth Douglas-fir stands nearer the coast (Hood and Morrison 1984). In plantations that replace these forests, many small disease centers arise, each consisting of only a few trees. All trees in every cluster over a wide area (more than 100 m across) are infected by a single *Armillaria* genotype, apparently derived from the colony of *A. ostoyae* that originally occupied the site (Adams 1974, Hood and Morrison 1984). By contrast, in selectively logged, old-growth, podocarp-hardwood rainforests in New Zealand, where *Armillaria* appears to be essentially non-parasitic, single-genotype colonies are small and densely distributed (Hood and Sandberg 1987). Disease centers in radiata pine plantations subsequently established on these sites are composed of different genotypes, some possibly originating from new introductions of basidiospore-derived material after clearfelling the natural forest (Benjamin and Newhook 1984a). In European forests, colonies of *Armillaria* species of one genotype seem to vary between about 10 m across up to 60 m *(A. ostoyae)*, or to around 200 m or more for *A. gallica* and *A. borealis* (Durrieu and Chaumeton 1988; *cf* Rishbeth 1972a, 1982, 1985b; Siepmann 1985; Siepmann and Leibiger 1989; Thompson and Boddy 1983).

Disease distribution is also affected by the distribution of stumps in the previous crop (Roth and others 1979, van der Pas 1981b). Many small, dead trees or stumps are more likely to ensure a widespread distribution of primary inoculum than are a few large ones (Pronos and Patton 1977), except when stumps are too small to act as effective inoculum.

Secondary Disease Spread

The primary inoculum eventually declines as a source of infection although the time required varies with stump size and host species. On some sites, hardwood stumps may act as inoculum for up to 30 years whereas conifers decompose more rapidly (Ivory 1987, Wingfield 1987). Whether the disease continues to spread through the plantation beyond the original infection center depends on whether or not infection is transmitted between healthy plants and adjacent infected plants of the same crop (the secondary inoculum). Secondary disease spread occurs in the same manner as primary spread (by root or rhizomorph contacts) and also by root grafting (Buckland 1953, Greig and Strouts 1983, Hintikka 1974, Peace 1962). It is limited by the distance between roots of neighboring trees and probably by the inoculum potential attained on infected hosts. Infected herbaceous plants, such as those found in vegetable or flower crops, are themselves unlikely to achieve sufficient inoculum potential for secondary disease spread; it is probable that only the initial, primary inoculum is functional in such plantings (Peace 1962, *cf* Wilson 1921, 1932). Rishbeth (1972b) suggested that very young, infected pine seedlings may also be too small to act as effective secondary inoculum, although apparent secondary spread has been observed among densely packed seedlings of radiata pine sown in nursery beds (pers. obs).

Van der Pas (1981b), working with radiata pine up to 5 years old in New Zealand, monitored mortality rates that followed van der Plank's (1963) model for disease increase without multiplication (slope of $\log_e [1/1-x]$ linear with time; x = proportion of dead trees) and

concluded no secondary spread happens in very young plantations (*cf* Whitney 1988b). On the other hand, Swift (1972) fitted mortality rates in up to 8-year-old slash pine in Zimbabwe to the model for disease increase by multiplication (slope of $\log_e [x/1-x]$ linear with time), implying that tree-to-tree spread of infection had occurred. Examining these results suggests that data from both authors may be used in either model with an acceptable degree of probability; conclusions based on this statistical method should be treated cautiously.

Other factors that govern the rate of spread of both secondary and primary disease are discussed in chapters 5, 8, 10 and 11. In addition, spread of disease may depend on the presence of susceptible carrier weed species (S. Singh 1978). Horner (1987) found that infection moved along kiwifruit roots faster than rhizomorphs grew through the soil. At times, these factors effect different rates of spread in different directions and thus distort shapes of disease foci. For example, infection frequently spreads along planting rows (fig. 9.6) giving rise to elongated disease centers (Horner 1985, Marsh 1952, Rishbeth 1978b, S. Singh 1978). Kable (1974) observed a directional trend toward irrigation channels in a peach orchard. Average rates of extension of disease centers are about 1 to 2 m per year (Chipompha 1987, Ivory 1987, Kable 1974, Piearce 1984, Podger and others 1978, Rishbeth 1980, Shaw and Roth 1976).

Subsequent Disease Development

Little is known about how infection centers behave in older forest plantations, but their boundaries might be expected to become more diffuse and irregular, and centers may merge and coalesce (McNamee and others 1989, P. Singh 1981c, Stage and others 1990). If *Armillaria* is widely distributed, the disease may affect randomly scattered trees rather than form discrete centers. MacKenzie (1987, *cf* Bloomberg and Morrison 1989) hypothesized a state of disease equilibrium in an older radiata pine plantation. He observed that although the percentage of basally infected trees remained fairly constant at 50-60% between ages 10 and 19 years, the root collars on 31% of the trees recovered from infection over this period while those on a slightly higher percentage of trees became newly infected. Chronically infected trees, often without crown symptoms (see chapter 5), have been reported in several forest plantations (Plavšić 1979, Rykowski 1980, Singh 1981c, Whitney and others 1989a); planted trees can resist and often recover from infection (Boullard and Gaudray 1975, Courtois 1979, Johnson and others 1972, Kawada and others 1962, Plavšić 1979). Observations in certain natural stands suggest that faster-growing trees may be more prone to chronic infection because their larger root systems are more likely to encounter the inoculum (Bloomberg and Morrison 1989, Hřib and others 1983).

Very little information describes how disease develops in successive crops planted on the same site except that *Armillaria* persists in subsequent rotations in both forest plantations and orchards (New Zealand For. Res. Inst. 1954; Delevoy 1946; Gibson 1957a,b, 1960, 1967; Holmsgaard and others 1961; Huntly and others 1961; Lundquist and Baxter 1985; Lysaght 1944; Millard 1949; Salmon and Ware 1937; Sisson and others 1978; Swift 1970; van der Pas 1981a). Knowledge is sparse partly because forest plantations which represent second or subsequent rotations are still uncommon and because in non-forest plantations the rotation status of the crop or stand is often unknown. Some authors considered that inoculum may dwindle and disappear after several rotations of conifer species (Gibson 1975, 1979; Peace 1962; S. Singh 1978; Wingfield 1987), but Redfern (1975) reported examples of disease in second- and third-rotation conifer crops following indigenous hardwoods. Inoculum may possibly increase in successive forest plantations, and Garrett (1956a) warned of a potentially greater need for eradication measures in planted stands than in natural forests. Multiple cropping may even introduce inoculum where it did not formerly exist (Delevoy 1946, Rishbeth 1978b). *Armillaria* currently inhabits certain second-rotation radiata pine stands in Kaingaroa Forest, New Zealand, on sites not formerly covered in indigenous forest (Gilmour 1954, MacKenzie and Self 1988, van der Pas 1981a). Planting young stock among established trees is another practice likely to enhance inoculum in diseased orchards (Kable 1974). This procedure, like multiple cropping, may also select for particular *Armillaria* species.

Stress and Predisposition

Disease development in plantations is influenced by two seemingly contradictory hypotheses of pathogen-host interaction often encountered in the literature. Some workers believe that *Armillaria* attacks secondarily or opportunistically (see chapter 7), being serious only on trees predisposed by various physical or biotic agents (Buckland 1953, Gremmen 1976, Huntly and others 1961, Johnson 1976, Sinclair and others 1987). Alternatively, attack may be primary; numerous examples in the literature confirm that vigorous plants may be directly infected (see chapter 6).

Whichever situation applies in a plantation probably depends on circumstances. *Armillaria* may be directly pathogenic on susceptible plant species but an opportunist on weakened, normally resistant hosts. Even so, it is not an easy matter to determine what is really occurring, due to the difficulty of deciding whether or not a host plant is actually under stress (Hiratsuka 1987, P. Singh 1980b). A major consideration is the pathogenicity of the *Armillaria* species concerned (see chapter 6; Guillaumin

and others 1984, 1989a; Rishbeth 1982, 1985b) and its inoculum potential (see chapter 4). Peace (1962) suggested that a three-way balance exists between the pathogen, the infected host, and the environment (*cf* Davidson and Rishbeth 1988, Sinclair and others 1987). Physiological host stress disturbs this balance in favor of the pathogen (Gibson and Goodchild 1961). The effect of stress on the host-pathogen interaction in both natural stands and plantations is discussed in chapter 7.

Disease Loss

Loss and Crop Age

In perennial plantations, the type and extent of disease loss is often closely related to the age of the crop (Rishbeth 1972a). In forest plantations, particularly those of conifers, mortality is the most common expression of disease early in the rotation since younger trees tend to be more susceptible and less tolerant of infection (Gibson 1975, Ivory 1987, Peace 1962, Sinclair and others 1987). In some stands, most mortality occurs during the first 8 years or so after planting (Bolland and Brown 1981, Fedorov and Poleschuk 1981, Fuller and James 1986, Longenecker and others 1975, Pronos and Patton 1977, Redfern 1978, Shaw and Calderon 1977, Usčuplić 1980, van der Pas 1981a), while in others mortality may continue for at least 25 years (Johnson and others 1972, Morrison 1981, Piearce 1984, Singh and Khan 1979, P. Singh 1981c). Comparatively early peak attack is also recorded in crop plants such as cinchona (Chevaugeon and Merny 1956), rubber (Anon. 1950, Pichel 1956), mulberry (G.-C. 1927), oil palm (Anon. 1948-1950, 1958), olive (Leach 1931), and tea (Gadd 1928-1930). However, in some plantation species, such as fruit trees (Hendrickson 1925, Kable 1974) and chestnuts (Bazzigher 1956), killing is less closely related to age. Marsh (1952) found older apple trees to be more susceptible in Great Britain.

Armillaria can kill even large specimens of some species; and significant losses may occur in older forest plantations or in urban plantings, particularly if trees are stressed (Greig and Strouts 1983; Kawada and others 1962; Mańka 1953, 1980, 1981; Moriondo 1981; Podger and others 1978). However, production losses in older stands are more often caused by growth reduction, butt rot, breakage, and windthrow, as a result of chronic infection (Dariichuk 1986 a,b).

Growth reduction due to *Armillaria* infection is rarely reported in annual crops (Conners 1936) and only occasionally in perennial horticultural plantations, possibly because reduced fruit yield is a more meaningful parameter of production loss (e.g., grape vines: Nieder 1980, Sisson and others 1978; kiwifruit: Horner 1985). Reports of growth reduction are more frequent from forest plantations (Peace 1962, Sinclair and others 1987, Williams and others 1989, *cf* Hřib and others 1983), but even in these crops, values are often presented only for tree height or stem diameter. Wood volume loss is rarely quantified (Morrison and others 1988, Shaw and Toes 1977, P. Singh 1980b, Terashita and others 1983). In older forest plantations, the increment of chronically infected trees can be depressed for extended periods although fluctuations may occur if circumstances change (Shaw and Toes 1977). By contrast, in acutely infected trees, which are usually relatively young, growth may drop sharply for 1-2 years prior to death (Lundquist 1988, Morrison 1981, Podger and others 1978, Szukiel 1980).

As trees of certain species become older, infection progresses from the roots to the lower stem heartwood. Butt rot caused by *Armillaria* is frequently present in older forest plantations, often associated with other decay fungi (Kató 1967b, Schönhar 1969, Storozhenko 1974, Yde-Andersen 1958, Zhukov 1968). Decay is also occasionally reported in other perennial crops such as fruit trees (Adaskaveg and Ogawa 1990, Guillaumin and others 1989b, Petersen 1960). The extent of rot depends on the host species (Greig and Strouts 1983, Peace 1962) and also on the species of *Armillaria*. In Britain, conifers are decayed mainly by *A. ostoyae*, *A. borealis*, and *A. cepistipes* whereas hardwoods are decayed by *A. gallica* (Gregory 1989, Gregory and Watling 1985, Rishbeth 1982).

Decay seldom extends more than a meter or so above ground level, depending on tree size, but it may be slightly more extensive in hardwoods than conifers (Greig and Strouts 1983). Even so, wood destruction represents volume loss from the more valuable butt log section, and the impact of this damage may therefore still be significant (Pegler and Gibson 1972). Losses also occur in butt rotted trees through stem breakage and windthrow (Greig and Strouts 1983, Ivory 1987, Moriondo 1981, Murray 1959, Sinclair and others 1987, Singh 1981c). The impact of windthrow and growth loss later in the rotation is probably more serious than that of early mortality since residual trees no longer balance the loss by compensatory growth during the remainder of the rotation (MacKenzie 1987). Butt rot also occurs in natural forests, and is discussed further in chapter 8 (which also includes examples from plantations, reference table 8.2).

Evaluation of Disease Impact

The overall economic loss caused by Armillaria root disease in plantations is rarely quantified effectively, probably due to the difficulties involved and the effort required. If attack is secondary, it is almost impossible to distinguish loss due to the predisposition stress from that caused by subsequent *Armillaria* infection (Chabro-

lin 1924, Hiratsuka 1987, Singh 1980b). For a complete and comprehensive economic evaluation, all aspects of disease loss, including uprooting, breakage, and less obvious effects of chronic infection such as growth loss and butt rot, must be considered.

Most disease impact assessments in forest plantations contain either qualitative comments such as "of no importance," "severe attack," "scattered mortality," or numerical estimates of the proportion of trees killed. Mortality is the most dramatic expression of the disease, and estimates have ranged from less than 3% (Morrison 1981, Singh and Khan 1979) to more than 50% (Ivory 1987, van der Pas 1981a). Mortality loss may be underestimated if counts are not made at regular intervals since small dead trees soon become lost among surrounding weed growth. On the other hand, Gibson (1979) suggested that the impact of mortality may be over-emphasized, at the expense of that due to chronic infection, because of its often spectacular appearance. Lower levels of mortality can represent a form of natural thinning, and are significantly compensated by increased growth of remaining trees (Courtois 1979, MacKenzie 1987). Other methods of impact assessment (see chapter 5) have been used, all of which to some extent underestimate the total economic loss. These methods include percentage of plantations or stands diseased in a forest, area or proportion of forest area out of production as a result of the formation of mortality gaps (Filip 1979, Jie 1982, Podger and others 1978, Redfern 1978, Shaw and Calderon 1977), and height (Singh 1981c) or diameter (Shaw and Toes 1977) increment reduction (see Lundquist 1988).

Occasional attempts have been made to assess the total loss throughout a rotation. Gibson (*in* Ofosu-Asiedu 1980) presented estimates of annual wood volume losses from conifer plantations in Malawi. Johnson and others (1972) judged that mortality gaps in plantations on Vancouver Island in British Columbia, Canada, were not of sufficient area to support a 48-year-old tree. They concluded the disease would have little impact by age 40-50 years as long as gaps did not expand and assuming that infected trees with healing stem lesions would recover. Shaw and Calderon (1977), and later MacKenzie (1987), evaluated the losses in a radiata pine stand in New Zealand. It was estimated that *Armillaria* reduced volume production by 6-13% in stands with a projected rotation length of 28 years (MacKenzie 1987).

Mortality more appropriately measures loss in orchards and horticultural crops than in forest plantations. Some reports containing estimates of disease impact in orchards or other non-forest crops are given in Division of Botany, Department of Agriculture (1923) and by Leefmans (1927), Zeller (1932), Pastore (1955), and Horner (1987). In contrast to forest plantations, it is normally economically feasible to replace diseased trees in orchards.

The impact of *Armillaria* in ornamental hosts varies greatly (Rhoads 1956). Losses are often high but difficult to express in economic terms because of the problem of assigning monetary values to plants grown for their aesthetic appeal. Costs can be quantified, however. These include control measures, removal of dead plants, stumps, and roots, site preparation or restoration, and the purchase of replacement plants. Although the expenses incurred by individual landowners are usually relatively low, the aggregate costs of Armillaria root disease in amenity plantings may be substantial.

Plantation Management and Disease

Control of *Armillaria* by reducing inoculum and other means is discussed in chapter 11. This section considers how routine tending procedures carried out in forest and horticultural plantations may indirectly influence disease severity, often without reducing the amount of inoculum. In practice, cultural management is rarely conducted specifically for disease control because such operations are costly and because reliable information on the expected economic gains is lacking (*cf* Pawsey and Rahman 1976a).

Planting

Young plants must always be considered vulnerable when exposed to a high inoculum potential. Using healthy, vigorous stock (Magnani 1978) and observing good planting practice (Birch 1937, Buckland 1953, Hadfield and others 1986, Johnson 1976, Ono 1970, Thies and Russell 1984) can minimize stress for trees planted on infected sites. The need for care at time of planting is supported by field observations (see also examples under "induced host stress"). Klomp and Hong (1985) found significantly higher *Armillaria* mortality among rooted radiata pine cuttings than among planted seedlings. They attributed this to better developed root systems on the latter. Hall and others (1971) observed the disease in planted seedlings whereas natural regeneration was unaffected. A number of authors have recommended using seed or container stock rather than bare-rooted plants in order to reduce plant stress (Hiratsuka 1987, Kessler and Möser 1974, Singh and Richardson 1973, Weissen 1981), but Shaw and Roth (1978) noted that other management considerations do not always permit this.

In theory, dense planting might be expected to favor disease spread due to competition stress and earlier root contact with adjacent plants. However, very little

field or experimental evidence documents the influence of planting density on disease (Hiley 1923). Pielou and Foster (1962) did not find a relationship between density and disease severity in Douglas-fir plantations, and attributed this to the fact that all the stands they examined were already old enough for root contact to have occurred.

Cultivation and Weed Control

Cultivation between rows of plants is a routine procedure in many orchards or planted crops. This practice controls weeds and improves soil texture, but it may also influence development of Armillaria root disease (Cutuli and Privitera 1986). Injuries sustained by crop plants during cultivation can cause stress and reduce disease resistance (Rosnev and Tsanova 1976). On the other hand, movement of infection across cultivated ground may be interrupted by the severing of roots. In a black currant plantation, Marsh (1952) observed a greater spread of disease along rows separated by parallel strips of cultivated ground than between rows. However, cultivation may also stimulate fresh growth from the cut ends of damaged rhizomorphs (Redfern 1973, Rykowski 1981c, Sewell 1965).

The amount of weed growth in a plantation is another factor that may influence disease development. Weeds may stress crop plants in young plantations through competition, especially in areas subject to droughts, rendering them more susceptible to infection, or damage if already infected. In addition, weeds may serve as bridges to promote disease spread between plants (Shaw and others 1976b, Singh and Bola 1981). However, using herbicides to kill weed growth or unwanted shade or shelterbelt trees may also increase disease severity by providing additional inoculum substrate (Andruszewska 1973, Boyd 1986, Pronos and Patton 1977, Schütt and others 1978). Cutting woody weed species may similarly enhance inoculum if root systems die and become colonized by the fungus. Application of certain herbicides may promote or inhibit the growth of *Armillaria* itself (Andruszewska 1973).

Thinning and Pruning

Little information describes how thinning impacts disease in forest plantations. Filip (1989a) found that thinning a number of conifer plantations had no significant effect on *Armillaria* mortality 5 years later.

Two factors in particular influence disease development when stands are thinned. Thinning may promote resistance to disease by reducing competition among residual trees (Johnson 1976, Singh 1981c, Williams and others 1989). Davidson and Rishbeth (1988) found that *A. mellea, A. ostoyae,* and *A. gallica* all caused extensive infections in oaks and pines weakened by crown suppression whereas only limited infections by *A. mellea* and *A. ostoyae* occurred in unsuppressed, subdominant oak and pine trees, respectively. On the other hand, thinning increases the amount of inoculum in a stand by providing fresh substrates for colonization, as noted earlier (refer under "disease establishment").

Thinning may affect diseased stands in other ways. It may lead indirectly to an unacceptably low stocking density if infected trees continue to die after the final thinning (Morrison 1981). Disease may be encouraged in final crop trees through stress from logging damage during commercial thinning (Johnson 1976).

The timing of thinning operations might be expected to influence the level of disease in plantations, but no information is available about the precise effect. Rishbeth (1978b) suggested that thinning late in the rotation may result in fewer, smaller, new disease centers, and so result in carryover of less inoculum into the subsequent rotation. Alternately, the bigger stumps created by late thinning may enable the fungus to develop a greater inoculum potential than on the smaller stumps from earlier thinnings. This could result in larger foci.

The stress of pruning live branches is likely to be harmful to plants already infected by *Armillaria*. However, the effects of pruning on the disease are even less studied than thinning. Chronically infected radiata pine trees were observed to die shortly after being pruned in a New Zealand stand (C.W. Barr, A. Zandvoort, pers. comm.). Excessive pruning of infected trees has also had serious effects in fruit orchards (Stahel 1950).

Fertilization

In many plantations, especially orchards and horticultural cultivations, application of fertilizers is an often routine part of management. Such treatment may especially benefit chronically infected plants stressed by nutrient deficiencies. However, other effects may also occur. Greater root growth may increase the chance of encounter with inoculum. Development of the inoculum itself may be promoted or discouraged by particular soil amendments (see chapter 4).

Fertilizer treatments have generally benefited diseased plants in several field trials, but definite results are not always observed, suggesting that complex interactions are involved. Singh (1983) showed that trees potted in a nutrient-rich soil (pH 4.8) were larger, became infected later, and had a smaller proportion of roots infected than plants in a soil deficient in certain nutrients (pH 3.8). Infection and mortality were lower in the fertile

soil and plants demonstrated active resistance by resin bleeding and callus formation. In a series of field trials in young pine plantations, Rykowski (1976b, 1980, 1981a, 1983) in many cases also demonstrated improved health to chronically infected trees after fertilizers were applied, although mortality rates were largely unaffected. Fertilizing appears to correct partially the tendency for root collar infection to hinder uptake of nitrogen and magnesium (Rykowski 1981b). Spurling and Spurling (1975) found that the damage caused by *Armillaria* in cultivated banana plants was reduced by applying potassium fertilizers. Clearly, additional trials are required before the effects of fertilization can be exploited in specific cases. Further work is also needed to clarify the effect of lime application on disease development (Anon. 1950, Pawsey and Rahman 1976a, Shields and Hobbs 1979, Sokolov 1971, van der Pas and Hood 1984).

Fertilizing with organic material is not always beneficial. Soil applications of processed urban refuse increased the incidence of disease in plantations established on infected sites, due apparently to host stress caused by toxic matter in the waste materials (Courtois 1973, Schwarz and Zundel 1975). Even so, Courtois (1979) found that trees which survived on sites treated in this way were larger than untreated plants. This was attributed either to the direct effect of the organic additive or to the "thinning" response among residual, surviving trees.

Control of Other Pests or Diseases

Trees with chronic *Armillaria* infection may succumb to other debilitating pest or disease agents present in plantations (see chapter 7). Relieving stress by routinely controlling these disease organisms or agents may promote resistance to *Armillaria*. Copper-based fungicide sprayed to control Dothistroma needle blight [*Dothistroma septospora* (Dorong.) Morelet (*D. pini* Hulbary)] in radiata pine stands in New Zealand reduced the impact of *Armillaria* in a chronically infected stand (Etheridge 1968, Shaw and Toes 1977).

Conclusions

Reports in the literature during the past 60 years indicate that species of *Armillaria* cause root disease in many planted hosts throughout the world. Attacks occur in softwood and hardwood forest plantations, woodlots, hedgerows, shelterbelts, orchards, and horticultural crops. The disease is also widespread in shade and amenity trees, ornamental shrubs, and herbaceous plants established in gardens, parks, and on roadsides. Records are particularly numerous from Europe, North America, Africa, and Australasia, but the disease is also present in the Soviet Union, Asia, and South America. In tropical parts of East Africa, Southeast Asia, and South America, it occurs in plantations at higher elevations where the climate is comparatively cool and moist.

Attack typically occurs in plants established on sites formerly occupied by forests or orchards, and in hosts interplanted among infected trees in existing stands. Infection spreads to new plants when roots encounter rhizomorphs growing from stump roots or when crop roots directly contact roots of colonized stumps. Plants tend to become infected in groups centered on stumps or root inoculum present in the soil, and deaths give rise to unstocked gaps. As the primary inoculum decays and becomes ineffective, disease centers may expand in perennial plantations through the creation of secondary inoculum in the crop itself. The rate of secondary spread between adjacent plants is governed mainly by host susceptibility, the degree of interaction between neighboring root systems, and the effectiveness of root-to-root transmission of infection. The subsequent development of disease centers in older plantations is not well understood.

Despite the merits of planting, and the obvious necessity of growing food crops and forest trees in plantations, several features of these production systems tend to encourage disease development when *Armillaria* is present. On previously wooded sites, inoculum increases to a high level early in the rotation when plants are young and especially vulnerable; new plants are predisposed to disease through transplant stress and the malformation of root systems; and spread of disease is favored by close spacing of even-aged stock. In monocultures the whole crop may be composed of a species susceptible to infection.

Mortality is the most obvious form of disease loss in young perennial plantations, while growth reduction, butt rot, lower stem breakage, and uprooting characterize chronic infection in older planted stands. Although often less spectacular than early mortality, chronic disease may have a more significant economic impact in forest plantations, but production loss has rarely been reliably quantified. The incidence of mortality is commonly quoted but has limited value unless disease centers are large, because increased growth of residual trees tends to compensate for earlier losses in tree numbers. The financial losses in ornamentals are difficult to quantify, and the cost of remedial measures may be underestimated in such plantings.

Disease development in infected plantations can be influenced by various management practices, such as the application of fertilizers, but choice of species, planting density, and the timing and intensity of thinning probably have the greatest effect in forest crops

(see chapter 11). Apart from using less susceptible species on infected sites, operations are rarely conducted specifically to ameliorate the impact of disease due to uncertainty about the effectiveness of such procedures and about their economic benefit. Further research is required to identify management regimes which will maximize returns from infected plantations.

TABLE 9.1 — *Armillaria* in planted conifer hosts, by country[1,2]

World zone	Continent	Country (and region)	References[2]
North Temperate	America (North)	Canada (General)	65. 2260
		Canada (BC)	41. 98; 42. 544; 57. 70; 63. 160; 73. 4051 (FA); 82. 7166 (FA); 85. 787; 86. 1121
		Canada (Ontario)	56. 799; 62. 418; 72. 1946; 73. 2753; 80. 941; 84. 374 (FA)
		Canada (Queb. & Maritime Provs.)	58. 380 (?), 684 (?); 62. 418; 63. 223; 67. 158b; 68. 1315; 71. 3387o; 86.312 (FA)
		Canada (Newf.)	63. 223; 70. 257a, 3032; 71. 3213; 74. 1670 (FA); 75. 958 (FA), 980 (AE); 79. 3084 (FA); 80. 5607 (FA); 82. 1632 (FA), 1969, 3079, 4411 (FA); 83. 10258 (SF); 84. 914
		USA (General)	86. 4102
		USA (Calif.)	48, 545 (ornamental?); 54. 79 (ornamental)
		USA (Wash., Ore., Idaho)	74. 6995 (FA), 7692 (FA); 76.420; 77. 4229; 80. 2375; 84. 2840 (FA)
		USA (New Mexico)	72. 1995
		USA (Minn., Wis.,Ind.)	28. 685; 60. 355; 77. 3733 (WA); 79. 3474; 83. 4468 (FA); 84. 5574 (FA)
		USA (Penn., NY, Conn.)	26. 394 (ornamental); 62. 259; 76.3873 (FA)
		USA (Georg., Fla.)	44. 504 (ornamental); 45. 435 (ornamental); 71. 1461; 73. 2051
	Asia	India (North)	56. 405; 80. 3431; 83. 4248 (FA)
		Japan	62. 551; 63. 499; 66. 635; 71. 2001
	Atlantic Europe (Western)	Portugal (Azores)	62. 487; 72. 4380; 77. 5835
		Belgium	29. 147; 36. 473; 46. 482; 49. 51; 50. 187; 82. 1971 (FA)
		Denmark	27. 447; 28. 351; 59. 633; 61. 497, 635; 62. 69
		Eire	40. 177; 45. 38
		Finland	40. 54; 47. 222 (ornamental?); 73. 1706; 75. 2738 (FA)
		France	23. 431; 27. 586; 33. 798; 67. 2120; 68. 90; 73. 880, 1290; 74. 6724 (FA); 77. 292 (FA); 82. 364 (FA), 1915 (FA), 4359; 86. 2211, 2438, 2441
		German Fed. Rep.	28. 349, 351; 31. 354, 698; 33. 739 (?); 38. 85; 40. 177; 52. 534; 55. 6, 760; 56. 800; 66. 2266; 68. 2882b; 70. 585, 2681; 73. 6898 (FA); 74. 220 (FA), 3031 (FA), 4396 (FA); 78. 3494 (FA); 82. 1368 (FA); 84. 875; 85. 3996
		Italy	50. 185, 188; 53. 460
		Netherlands	40. 195; 58. 745
		Norway	51. 202; 66. 2978; 80. 4799
		Sweden	38. 752 (ornamental)
		Switzerland	28. 351; 33. 195; 54. 570; 55. 328; 57. 69; 58. 560
		United Kingdom	27. 197, 447; 29. 78; 33. 130; 35. 803; 38. 715; 40. 405, 506; 46. 428; 51. 591; 57. 601 (orna mental); 59. 714; 62. 262; 63. 349; 64. 1164, 3023; 66. 888; 67. 3213; 68. 2564; 71. 2000; 72. 4375d; 73. 3431; 75. 2125 (FA); 76. 6820 (FA); 78. 4869 (FA); 79. 3491; 80. 5923; 82. 5974; 85. 2171; 86. 381, 2440; 88. 3140

TABLE 9.1 — *(Continued)*

World zone	Continent	Country (and region)	References[2]
	Europe (Eastern)	Czechoslovakia	27. 213; 28. 351; 45. 257; 76. 2984 (FA); 79. 856 (FA); 83. 1921 (FA); 84. 935; 87. 5207 (FA)
		German Dem. Rep.	28. 351; 31. 699; 32. 141; 33. 480, 739 (?); 37. 647; 75. 4853 (FA)
		Poland	26. 714; 45. 257; 53. 44; 54. 391; 55. 499; 62. 552; 64. 2416, 2433; 69. 936; 73. 3875 (FA); 74. 3001 (FA), 5258 (FA), 7504 (FA); 75. 1556 (FA), 7809 (FA); 78. 2234 (AE); 81. 730 (FA), 3032 (FA), 4126 (FA); 82. 381 (FA), 419, 2273 (FA), 3631 (FA); 84. 919; 85. 3770 (FA), 4827 (FA), 6070 (FA); 86. 5200; 87. 1573 (FA), 1893 (FA)
		Rumania	72. 3608
		Yugoslavia	76. 4309 (FA), 4312; 81. 6678; 82. 1632 (FA)
	USSR	Soviet Union (western, incl.Belorussia, Ukraine, Caucasus)	28. 415; 65. 1283; 66. 1528; 75. 6374 (FA); 81. 4516 (FA), 6092; 86.3004; 87. 2082, 2411 (FA), 4716 (FA)
		Soviet Union (Urals, Krasnoyarsk)	62. 417; 65. 1281 (?), 1978 (?)
South Temperate	Africa	South Africa	33. 142; 34. 425; 37. 355, 784; 54. 657; 56. 405; 81. 2785; 83. 2174; 88. 1500
	America (South)	Brazil (southern)	64. 2095; 67. 2848
		Chile	63. 350; 65. 552; 67. 3227
	Australasia	Australia	23. 298; 71. 3384f; 82. 1632 (FA)
		New Zealand	34. 533; 38. 714; 45. 297; 53. 52; 54. 328, 329; 55. 267; 56. 565; 63. 156, 635; 69. 618c; 74. 2453 (FA); 82. 2277 (FA); 85. 2889 (FA)
Tropics	Africa	General	82. 1634 (FA)
		Kenya	51. 141, 309; 54. 16; 58. 116, 190; 60. 509=61. 569; 61. 436; 65. 2615a
		Malawi	49. 200; 53. 669
		Mauritius	46. 52
		Tanzania	52. 225; 55. 350; 65. 1924, 2615a
		Zimbabwe	62. 762; 63. 727; 68. 3020; 72. 4431
	America (Central) /Caribbean	Jamaica	68. 2958a
	America (South)	Peru	78. 3635
	Oceania	Fiji	65. 1351b
		USA (Hawaii)	65. 849 (FA); 74. 6994 (FA)

[1]Published reports of *Armillaria* attack in coniferous plantation forests (with occasional records for ornamental plantings). All listings (except two) refer to Northern Hemisphere host genera, including tropical pines: *Abies, Cedrus, Chamaecyparis, Cryptomeria, Cupressus, Juniperus, Larix, Metasequoia, Picea, Pinus, Pseudotsuga, Thuja, Tsuga* (exceptions:51.141 for Kenya and 53. 669 for Malawi, concerning *Araucaria, Callitris, Widdringtonia*). References to trees attacked within their natural distribution ranges (mainly North Temperate) are sometimes doubtfully included; it is not always clear whether these are planted or naturally seeded.

[2]Sources: Review of Plant Pathology (Review of Applied Mycology), unless otherwise stated.
Code: year (**not** volume No.). No. of abstract or [prior to 1964] page (abstract journal title abbreviation, as applicable).
AE, Review of Applied Entomology, Ser. A; FA, Forestry Abstracts; hA, Helminthological Abstracts, Ser. B; HA, Horticultural Abstracts; PB, Plant Breeding Abstracts; SF, Soils and Fertilizers; WA, Weed Abstracts.
Compilation: 1922-1972, manual search (keywords: ARMILLARIA, CLITOCYBE TABESCENS)
1972-June 1988, computer search (descriptor: ARMILLARIA ( ) MELLEA; Commonwealth Agricultural Bureaux Dialog Information Retrieval Service; duplicate reports not listed).

Table 9.2a — *Armillaria* in planted, non-conifer (angiosperm) hosts, by country.(a) Species used in commercial forestry, for shelter, or as ornamentals[1,2]

Host group	World zone	Continent	Country (and region)	References[2]
Northern deciduous broadleaf trees (e.g., beech, *Fagus* birch, *Betula;* chestnut, *Castanea;* elm, *Ulmus;* oak, *Quercus;* poplar, *Populus;* willow, *Salix*)	North Temperate	America (North)	Canada (Ont.)	64. 1755 (?)
			Canada (Queb., Newf.)	62. 418(?); 74. 1669 (?;FA); 83. 4775
			USA (Calif.)	59. 178; 71. 3187
			USA (Central & East)	32. 411; 41. 183; 42. 272; 76. 7439 (AE); 87. 78
			USA (Mississ., Fla.)	44. 417; 51. 1332 (?;FA);70. 864
		Asia	Chinese Peoples' Rep.	47. 421
			Japan	60. 354
			Pakistan	88. 5414 (FA)
		Europe (Western)	Belgium	73. 6416 (FA)
			France	23. 431; 24. 5, 8; 27. 586; 28. 290; 45. 436; 47. 320; 66. 626; 82. 364 (FA); 84. 3385 (FA), 6619 (FA); 85. 5785 (FA); 86. 2211, 2438 , 2441
			German Fed. Rep.	70. 1156; 72. 3694k
			Italy	54. 568; 61. 493; 64. 3033; 79. 1785 (FA); 81.4684
			Netherlands	31. 696; 40. 195
			Spain	64. 846
			Switzerland	57. 69 (?), 673; 61. 492
			United Kingdom	24. 244; 27. 198; 28. 126; 46. 428; 48. 399; 70. 1157; 72. 2871b; 74. 7708 (FA); 79. 899; 82. 1632 (FA), 5974; 83. 4450; 85. 2171; 86. 381, 2440
		Europe (Eastern)	Bulgaria	81. 3154 (FA)
			Hungary	86. 781
			Poland	74. 1768 (FA); 81. 3032 (FA); 87. 2326 (FA)
			Yugoslavia	26. 705 (?); 27. 5 (?); 30. 278
		USSR	Soviet Union (western, Novosibirsk)	59. 426 (?); 66. 1528 (?); 69. 2009
	South Temperate	Africa	South Africa	27. 237
		Australasia	Australia	55. 285
Other hardwood trees (e.g., *Acacia; Casuarina; Eucalyptus; Grevillea; Leucaena;* teak, *Tectona; Terminalia*)	North Temperate	Africa	Tunisia	71. 285, 1396
		America (North)	USA (Calif.)	28. 494; 71. 3187 (?); 80. 500 (FA)
			USA (Fla.)	30. 159; 41. 563; 42. 486, 497; 44. 417, 504; 48. 279; 51. 1332 (FA); 53. 406; 57. 560
		Europe (Western)	German Fed. Rep.	70. 1156 (?)
		Europe (Eastern)	Hungary	86. 781
	South Temperate	Africa	South Africa	33. 142; 37. 784; 88. 1500
		Australasia	Australia	59. 564 (?);77. 3 (hA; ?);82. 1632 (FA)
	Tropics	Africa	Ghana	27. 19
			Kenya	51. 141; 60. 214, 509; 61. 569; 82. 7157 (FA)
			Madagascar	55. 107, 451
			Malawi	29. 202; 40. 311; 53. 669
			Mauritius	30. 507; 52. 537; 60. 633

Table 9.2a — *(Continued)*

Host group	World zone	Continent	Country (and region)	References[2]
			Tanzania	33. 201; 36. 746 (?); 52. 225; 67. 1348
			Uganda	24. 509; 27. 15
			Zaire	51. 512
			Zimbabwe	62. 126, 761; 63. 727
		America (South)	Peru	78. 3635
		Asia	India (south)	60. 123; 64. 558
			Indonesia (Sumatra, Java, Sulawesi)	25. 79; 31. 525
			Sri Lanka	28. 745; 29. 470; 31. 275
Shrubs and ornamental herbs	North Temperate	America (North)	USA (General)	33. 696
			USA (Wash., Ore.)	32. 786; 48. 134; 70.1677
			USA (Calif.)	54. 79; 59. 147; 68. 2734; 69. 874; 72. 1551; 80. 1616
			USA (Fla.)	42. 497; 44. 504; 48. 23; 51. 1332 (FA); 57. 560
		Europe (Western)	France	72. 4086; 82 4011; 86. 2211, 2438
			German Fed. Rep.	70. 1156
			Italy	86. 4983
			Netherlands	40. 195
			Switzerland	57. 69
			United Kingdom (incl. Jersey)	29. 628; 32. 376; 35. 366; 36. 478; 38. 823; 48. 462; 53. 332; 54. 484, 605; 61. 267; 64. 2792; 85. 2211; 86. 381
		Europe (Eastern)	Czechoslovakia	70. 2560
		USSR	Soviet Union (Georgia)	67. 643
	South Temperate	Africa	South Africa	27. 237
		Australasia	Australia	48. 275; 59. 564; 67. 660
	Tropics	Africa	Malawi	34. 216; 35. 834; 36. 780
			Tanzania	48. 158
			Zaire	51. 196
			Zimbabwe	62. 126; 65. 598
		Asia	India (south)	64. 558

[1]Published reports of *Armillaria* attack in forest plantations, woodlots, gardens, parks, roadsides, hedgerows, and farm shelterbelts. Includes ornamental nursery plants, trees established for shade or edible fruit supply (home or local, non-commercial use), and plants used to shelter production crops or provide green manure. References to trees attacked within their natural distribution ranges (mainly North Temperate) are sometimes doubtfully included; it is not always clear whether these are planted or naturally seeded.

[2]As for Table 9.1.

TABLE 9.2b — *Armillaria* in planted non-coniferous (angiosperm) hosts, by country. (b) Species used in economic production (except forestry)[1,2]

Crop	World zone	Continent	Country (and region)	References[2]
Avocado *(Persea)*	North Temperate	America (North)	USA (Calif.)	35. 707; 49. 630 (?); 56. 907; 66. 1868
	Tropics	America (South)	Ecuador	60. 435
Banana	North Temperate	America (North)	USA (Fla.)	32. 382; 42. 497
	South Temperate	Africa	South Africa	72. 1693
		Australasia	Australia	34. 356; 67. 1674 (?)
	Tropics	Africa	Kenya	54. 141; 65. 2697a
			Malawi	76. 5091 (HA)
			Tanzania	33. 552; 53. 324
			Zimbabwe	62. 126
Berryfruit – cane (*Ribes*; currant, gooseberry)	North Temperate	America (North)	Canada (BC)	38. 49
			USA (Wash., Ore.)	23. 278; 44. 112; 45. 423
– bramble (*Rubus*; blackberry, loganberry, raspberry)			USA (Calif.)	52. 24
		Europe (Western)	United Kingdom	24. 525; 53. 320
		USSR	Soviet Union (Krasnodar)	72. 4191
	South Temperate	Australasia	Australia	49. 528; 60. 327
			New Zealand	42. 29
Cacao (cocoa)	Tropics	Africa	Cameroon	57. 383
			Ghana & Togo	24. 325; 25. 463; 27. 19, 659, 704; 28. 93, 565
			Ivory Coast	36. 16
			Madagascar	55. 107
			Nigeria	69. 755c
			Sao Tome & Principe	26. 149; 80. 5680
			Uganda	24. 509; 26. 17; 74. 1931 (HA)
			Zaire	47. 235; 49. 31, 272
		America (Central)	Mexico	49. 328
		America (South)	Brazil	39. 93 (?)
			Colombia	60. 15
		Australasia	Papua New Guinea	52. 9
Cactus (*Opuntia ficus-indica;* edible fruit)	North Temperate	Europe (Western)	Italy (Sicily)	83. 3151
Cassava *(Manihot)*	Tropics	Africa	Tanzania	33. 552
			Zaire	57. 308
		America (South)	Brazil	36. 278 (?)
Chesnut *(Castanea)*				Refer Table 9.2 (a)
Cinchona (quinine)	Tropics	Africa	Guinea	59. 94
			Zaire	44. 431; 46. 45, 154; 51. 196
		America (South)	Peru	55. 675
		Asia	Indonesia (Java, Sumatra)	23. 9; 24. 189; 25. 79; 28. 308; 30. 161; 31. 298; 37. 160; 39. 578
Citrus fruit (grapefruit, lemon, lime, orange, tangerine etc.)	North Temperate	Africa	Libya	60. 658
			Morocco	64. 3222
			Tunisia	33. 302
		America (North)	USA (Calif.)	26. 358; 30. 766; 32. 40; 41. 160, 360; 45. 225= 366; 49. 630; 51.

TABLE 9.2b — *(Continued)*

Crop	World zone	Continent	Country (and region)	References[2]
				608; 54. 79; 55. 366, 641; 64. 520; 68. 1886; 69. 469; 70. 3661; 71. 1224d; 74. 2805 (HA); 77. 1943; 80. 5143, 5144
			USA (Fla.)	31. 99; 32. 365; 42. 486, 497; 48. 279; 72. 1495
		Europe (Western)	Cyprus	32. 696
			France (incl. Corsica)	56. 447; 86. 719
			Greece (Crete)	39. 672
			Italy (incl. Sicily)	36. 213; 85. 4323; 88. 196
			Malta	34. 81; 35. 618; 36.780
			United Kingdom	81. 6692 (HA)
		Europe (Eastern)	Yugoslavia	55. 297
	South Temperate	Australasia	Australia	23. 354; 27. 101; 33. 142; 36. 280; 37. 451; 42. 440; 44. 296; 46. 558; 47. 74, 337; 49. 453; 53. 175; 62. 151
	Tropics	Africa	Kenya	85. 2544
			Malawi	33. 10
		Asia	Indonesia (Java)	38. 162; 39. 794; 40. 143
Coffee	Tropics	Africa	General	37. 454
			Cameroon	55. 86
			Central African Rep.	54. 537
			Ethiopia	67. 329; 69. 5; 70. 918
			Guinea	59. 94; 63. 612
			Ivory Coast	50. 99
			Kenya	26. 299 (?); 30. 31 (?); 60. 228; 73. 2278
			Madagascar	32. 699 (?); 34. 506 (?); 55. 107, 451; 58. 408; 63. 683; 64. 2914
			Malawi	28. 239; 62. 7
			Mauritius	72. 8a
			Sao Tome & Principe	59. 745 (?); 80. 4618
			Tanzania	33. 201; 34. 114; 36. 261; 51. 509
			Uganda	23. 409; 24. 509; 28. 701; 33. 422
			Zimbabwe	62. 761
		Asia	Indonesia (Java)	39. 452
		Australasia	Papua New Guinea	56. 423
Cola acuminata (edible nut)	Tropics	Africa	Ghana	27. 19
Cork *(Quercus suber)*	North Temperate	Europe (Western)	France	23. 431
			Italy (Sardinia)	64. 845
			Portugal	72. 4380 (?)
Cotton	Tropics	Africa	Zaire	49. 31
Fig *(Ficus carica)*	North Temperate	Africa	Algeria, Morocco, Tunisia	24. 88
		America (North)	USA (Calif.)	26. 37 (?); 48. 372
		Europe (Western)	France	47. 328; 82. 4011; 86. 2438, 5626
Flower production	North Temperate	Europe (Western)	France	Refer Table 9.2 (a)
Geranium/Pelargonium (oil source; see also Table 9.2a, Zaire)	Tropics	Africa	Kenya	56. 358
			Tanzania	51. 79
General	Tropics	Africa	Tanzania	36. 746
Grapevine *(Vitis)*	North Temperate	America (North)	USA (Fla., Missouri)	25. 585; 27. 460
			USA (Calif.)	51. 402; 64. 520; 73. 7573 (HA); 80. 1097 (SF), 9620 (PB)

TABLE 9.2b — (*Continued*)

Crop	World zone	Continent	Country (and region)	References[2]
		Europe (Western)	Austria	81. 1114 (HA)
			Belgium	57. 86; 64. 1512v
			France	25. 525 (?); 27. 563, 586; 30. 360 (?); 38. 653; 59. 118 (?); 60. 767; 61. 507; 82. 4011, 4359; 86. 2211, 2438, 2441
			Greece	55. 706
			Italy	36. 774; 46. 436; 56. 658
			Spain	34. 745; 36. 75, 541; 53. 419
			Switzerland	56. 415; 76. 3131 (HA)
		Europe (Eastern)	Bulgaria	25. 460
		USSR	Soviet Union (western, Georgia)	64. 1535m; 77. 3152
	South Temperate	America (South)	Brazil (southern)	83. 3159
		Australasia	Australia	27. 101
Guava *(Psidium)*	North Temperate	America (North)	USA (Fla.)	30. 159; 42. 497
	Tropics	Africa	Malawi	66. 700d
Hazelnut *(Corylus)*	North Temperate	America (North)	USA (Oreg.)	48. 165
		Europe (Western)	Italy	85. 4520
			United Kingdom	26. 278
Hops *(Humulus)*	North Temperate	America (North)	USA (Oreg.)	49. 593
		Europe (Western)	Greece	60. 210
			United Kingdom	36. 478, 605; 37. 822; 40. 364; 43. 39; 44. 2
	South Temperate	Australasia	Australia	60. 327; 63. 137
Hydnocarpus anthelmintica (medicinal oil)	Tropics	Africa	Zaire	49. 271
Kiwifruit *(Actinidia)*	North Temperate	America (North)	USA (Calif.)	72. 2721
		Europe (Western)	France	86. 2438 (?)
	South Temperate	Australasia	New Zealand	71. 3066
Lavender (*Lavendula*; perfume)	North Temperate	Europe (Western)	France	34. 99
			United Kingdom	39. 724
Litchi (*Litchi*; edible fruit)	North Temperate	America (North)	USA (Fla.)	42. 497 (?); 56. 781; 62. 618 (?)
Loquat *(Eriobotrya)*	North Temperate	America (North)	USA (Calif.)	27. 175; 49. 630 (?)
	Tropics	Africa	Tanzania	38. 15
Macadamia Nut	Tropics	Africa	Zimbabwe	68. 2588
Mangel (*Beta*; cattle food)	North Temperate	America (North)	Canada (New Bruns.)	36. 632
Mango *(Mangifera)*	Tropics	Africa	Ghana, Uganda, Zimbabwe	Refer Table 9.2 (a)
Mulberry *(Morus)*	North Temperate	Europe (Western)	France	25. 201; 27. 567
			Italy	29. 207; 30. 213
		Europe (Eastern)	Hungary	86. 781
		USSR	Soviet Union (Uzbek.,	46. 17

TABLE 9.2b — *(Continued)*

Crop	World zone	Continent	Country (and region)	References[2]
			Kirghiz., Tadzhik. ?)	
	South Temperate	Australasia	Australia	27. 101
Oil palm *(Elaeis guineensis)*	Tropics	Africa	Zaire	47. 235; 49. 31, 272; 50. 236, 292; 51. 155, 512; 57. 513
Olive	North Temperate	Europe (Western)	France	24. 348; 82. 4011; 86. 1420, 2438
			Italy	46. 492; 51. 235; 56. 658; 62. 358; 76. 1641 (HA)
			Spain	46. 508
	Tropics	Africa	Malawi	31. 707
Papaya (papaw)	Tropics	Africa	Kenya	48. 483; 71. 5b
			Tanzania	48. 483; 50. 145
Passiflora spp. (passionfruit, granadilla)	South Temperate	Australasia	Australia	27. 101
	Tropics	Africa	Zimbabwe	65. 598
Pecan *(Carya illinoensis)*	North Temperate	America (North)	USA (Georgia)	71. 294
Persimmon *(Diospyros)*	North Temperate	America (North)	USA (Calif.)	48. 372 (?); 49. 630 (?)
Pome fruit *(Malus, Pyrus;* apples, pears)	North Temperate	America (North)	Canada (BC)	23. 304; 26. 146
			USA (Wash., Ore.)	24. 89; 26. 746; 30. 212
			USA (Calif.)	23. 394; 32. 40; 25. 21; 26. 37; 34. 552; 40. 24; 48. 372; 64. 520 (?), 2855; 72. 1631
			USA (Louis., Fla.)	41. 169; 42 497
		Europe (Western)	France	23. 431 (?); 59. 125; 86. 2438
			German Fed. Rep.	35. 677 (?); 71. 1867 (?)
			Italy	74. 2033 (HA)
			Malta	36. 780; 38. 589
			Netherlands	51. 420
			Spain	34. 745 (?); 36. 75 (?), 541 (?); 42. 338
			Switzerland	50. 443
			United Kingdom	24. 259; 26. 278; 37. 822; 53. 320; 54. 91; 60. 178 (?); 70. 1426
	South Temperate	Africa	South Africa	37. 784 (?)
		America (South)	Chile	69. 2282
		Australasia	Australia	23. 353; 25. 399 (?); 26. 105; 27. 101; 28. 304 (?); 35. 451 (?); 45. 318 (?); 56. 613 (?); 60. 327; 62. 158; 67. 2775
	Tropics	Africa	Kenya	51. 309
			Tanzania	48. 224
			Zaire	51. 196
			Zimbabwe	36. 705; 38. 46; 66. 2a
Rubber *(Hevea)*	Tropics	Africa	Cameroon	66. 1166
			Central African Rep., Chad, Congo &/or Gabon	53. 507
			Congo	64. 3003
			Nigeria	64. 3003, 3172; 66. 1166
			Uganda	24. 509
			Zaire	47. 466; 48. 123, 382; 49. 271; 50. 236, 292; 51. 512; 54. 413; 55. 583; 56. 925; 57. 308; 61. 486

TABLE 9.2b — *(Continued)*

Crop	World zone	Continent	Country (and region)	References[2]
Stone fruit (*Prunus*; almond, apricot, cherry, peach, plum, etc.)	North Temperate	America (North)	Canada (BC, Ont., Queb.)	26. 146; 47. 429 (?); 75. 2151 (HA)
			USA (Wash., Ore.)	26. 746
			USA (Calif.)	25. 681; 26. 37; 31. 323; 34. 552; 36. 518; 40. 24; 41. 161; 45. 453; 46. 446; 48. 372; 49. 630; 52. 68; 53. 24; 54. 36; 64. 520 (?), 2855; 72. 4174; 77. 1943
			USA (Wisc., Ill., Mich.)	23. 481; 54. 733; 88. 4191 (FA)
			USA (Mary., Nth Carol., Sth Carol., Georg., Fla.)	30. 159; 41. 68, 562; 42. 497; 44. 25; 53. 682; 54. 434; 55. 603; 61. 370; 62. 398; 63. 618; 69. 1842
		Europe (Western)	France	23. 431 (?); 26. 304; 27. 563 (?); 30. 116, 465; 32. 791; 49. 527; 50. 265; 54. 161; 56. 109, 305; 82. 4011, 4359; 86. 2211, 2438, 2441; 87. 9220 (HA)
			German Fed. Rep.	35. 677 (?); 71. 1867 (?); 73. 63 (HA)
			Italy	34. 706; 56. 658; 63. 562; 68. 2215
			Netherlands	48. 244
			Spain	34. 745 (?); 36. 75 (?), 541 (?)
			United Kingdom	28. 647; 37. 822; 60. 178 (?)
		Europe (Eastern)	Hungary	47. 344; 48. 140; 86. 781
			Yugoslavia	86. 808
	South Temperate	Africa	South Africa	27. 237; 37. 784 (?)
		Australasia	Australia	25. 399 (?); 28. 304 (?); 35. 451 (?); 45. 318 (?); 47. 285; 48. 27; 56. 613 (?); 65. 1354g; 67. 2775; 73. 2099f
	Tropics	Africa	Kenya	37. 796 (?); 51. 309; 58. 136
			Zimbabwe	36. 705; 38. 160
Strawberry (*Fragaria*)	North Temperate	America (North)	USA (Wash., Ore.)	29. 727; 31. 163; 32. 727; 39. 402; 45. 423
			USA (Calif.)	61. 617
		Europe (Western)	United Kingdom	27. 650
Sugar cane (*Saccharum*)	Tropics	Africa	Tanzania	33. 552
Tea	North Temperate	Asia	India (northeast)	40. 369; 83. 3975; 85. 2155
	Tropics	Africa	Kenya	53. 513 (?); 58. 512; 60. 214; 61. 73, 723; 76. 10851 (HA)
			Malawi	28. 275; 29. 202; 33. 10; 34. 216; 35. 14; 36. 780; 37. 209, 564; 40. 311; 49. 200; 74. 8119 (HA); 81. 3935
			Mauritius	52. 538
			Mozambique	50. 89
			Tanzania	36. 261; 53. 513 (?); 55. 350
			Uganda	24. 509; 29. 756; 37. 838
			Zaire	49. 272; 57. 309 (?); 60. 440
			Zimbabwe	59. 295; 65. 598
		Asia	India (south)	55. 489; 60. 123; 64. 558; 66. 595b; 76. 8835 (HA)
			Indonesia (Java, Sumatra)	23. 9; 24. 5, 64, 611; 26. 585; 31. 409; 38. 162, 202; 39. 579
			Malaysia	37. 657 (?)
			Sri Lanka	23. 295; 28. 745; 29. 469; 31. 275; 40. 678 (?)

TABLE 9.2b — (Continued)

Crop	World zone	Continent	Country (and region)	References[2]
		Australasia	Papua New Guinea	56. 423
Tung (*Aleurites;* oil)	North Temperate	America (North)	USA (Louis., Fla.)	37. 426 (?); 41. 169; 44. 504; 49. 365
	Tropics	Africa	Malawi	40. 626; 49. 200; 51. 295; 53. 703; 62. 734
		Asia	India	50. 588 (?; see 55. 267)
Vegetables (carrot, parsnip, potato; also tomato; see elsewhere for cassava, mangel)	North Temperate	America (North)	Canada (BC)	37. 832
		Europe (Western)	Belgium	39. 724; 71. 460b
			United Kingdom	22. 357; 48. 462
		USSR	Soviet Union (Leningrad)	46. 316
	South Temperate	Australasia	Australia	27. 101; 33. 142; 34. 257; 37. 118
Walnut (*Juglans*)	North Temperate	America (North)	USA (Ore.)	24. 89; 42. 310; 49. 94; 51. 594; 52. 463; 59. 39
			USA (Calif.)	26. 37; 34. 552; 45. 453; 48. 103, 372
		Europe (Western)	France	22. 35, 77; 24. 179; 25. 201, 577; 26. 526; 27. 200, 426, 563, 586; 28. 686; 32. 95; 34. 366 (?); 36. 763; 72. 4403 (?); 84.4723 (FA)
			Italy	46. 427
		Europe (Eastern)	Bulgaria	76. 6817 (FA); 77. 871 (FA), 4716
			Czechoslovakia	27. 6
			Hungary	48. 140; 86. 781
	South Temperate	Australasia	Australia	54. 610

[1]Published reports of *Armillaria* attack to planted trees, shrubs, or herbaceous species used as commercial food crops or for processed products (except timber or pulpwood).

[2]As for Table 9.1.

CHAPTER 10

Modeling the Dynamics, Behavior, and Impact of Armillaria Root Disease

Charles G. Shaw III, Albert R. Stage, and Peter McNamee

Information on the ecological, biological, and pathological attributes of *Armillaria* spp. and the root disease they cause comprises the major portion of this book. Integration of this material, particularly as it relates to the portrayal of disease dynamics and the quantification of disease impacts, would markedly enhance its utility for foresters, orchard managers, and scientists. Models can accomplish this objective, and some have been developed for root disease caused by *Heterobasidion annosum* (Fr.) Bref. (Alexander and others 1985, Pratt and others 1989) and *Phellinus weirii* (Murr.) Gilbn. (Bloomberg 1988).

This chapter describes how information on Armillaria root disease has been used to develop a predictive model of disease dynamics, behavior, and impact. Foresters currently make decisions about root disease management using their mental model of the disease process in the affected area as a guide to select treatment alternatives for the land. The process of building a predictive model combines existing data and the key features of the mental models of several knowledgeable forest managers and scientists in a set of mathematical equations. By pooling and structuring the knowledge of many, we should have a better model for individual stand management as well as for overall forest planning than would be assembled by any single manager or scientist. An important benefit of the model-building process is a highlighting of our still inadequate understanding of many biological aspects of Armillaria root disease, increased knowledge of which is necessary to improve management.

Pathologists have a wealth of information about *Armillaria* spp. and the root disease they cause. However, even when these data are published they frequently are not available to managers in a form that directly assists decision making. Because pathologists have the best biological understanding of root disease dynamics as well as the limitations of available data, it is imperative that they define the biological assumptions necessary to develop a predictive model. If research pathologists diligently perform this role, then the resulting model not only becomes a tool of immediate use to managers, but also becomes a quantitative description of a series of hypotheses about root disease dynamics, behavior, and impact. As such, it can aid scientists in identifying serious data gaps and thus help to define and prioritize research needs.

Scientists trained in the research process may find development of a management-oriented model troublesome because professional judgement, rather than statistically analyzed data, often becomes the only, or at least primary, basis for a generalized assumption that can markedly influence the outcome of a model prediction. The traditional researcher is far more comfortable with the model of a scientific paradigm where behavior is judged at levels that are often far removed from the decision criteria that are required of a predictive model for management. Consequently, knowledge gaps can be left to future research as no immediate opportunity exists for application of the model. Forest managers who routinely encounter stands severely impacted by root diseases are desperate for tools to deal with these complex and damaging problems. Thus scientists, who best understand these problems, even if that understanding comes primarily from their professional judgements and experiences, can no longer take a laissez faire, hands-off approach to management-oriented modeling.

This chapter describes the integration of our current understanding of Armillaria root disease dynamics and the damage the disease causes in various conifer ecosystems in western North America (see chapter 8) into a predictive model for management use in silviculture and forest planning (Stage and others 1990). The hypotheses or assumptions that underlie the quantitative relationships contained in the model are discussed and referenced to information presented elsewhere in this book. In addition, direction is provided to indicate how model users (both managers and scientists) can examine alternative hypotheses about the dynamics and behavior of Armillaria root disease.

The process used to build the Western Root Disease Model (Brookes 1985, Eav and Shaw 1987, Shaw and others 1985) can serve as a prototype for modeling the dynamics and behavior of Armillaria root disease in other forest ecosystems or orchards.

History and Structure of the Western Root Disease Model

Recognizing the serious economic impact of annually losing 6.8 million cubic meters of timber in the Western United States to root diseases (Smith 1984), the USDA Forest Service initiated a project to develop a root disease model (Brookes 1985). The protocols of Adaptive Environmental Assessment, as outlined by Holling (1978), were used to develop the Western Root Disease Model. In this procedure, serial workshops allow various experts in disease recognition, biology, and management to meet with potential model users for short periods of intense interaction. Through the direction and assistance of model coordinators, they develop a conceptual model of the problem and possible management actions to mitigate damaging effects (Brookes 1985). The coordinator is then responsible for converting this information into a working, predictive model that is further refined at subsequent workshops through additional input from specialists and potential users. The process itself is not new, but it creatively extends the scientific method from the individual investigator to a corporate surrogate (Walters 1986).

A recognized strength of the procedure is that it gives ownership of the final product, and thus a desire to have a quality item produced in a timely manner, to all who were involved with its development. Also, because the model building is cooperative, scientists can appreciate the need to provide managers with the best current understanding of root disease spread and impact, and managers can recognize the critical uncertainties in our knowledge of root disease biology and the need for further research.

The model was developed as a tool to aid foresters with overall, forest-level planning and with the design of silvicultural treatments in individual stands affected by root disease. The model can project the effects of various levels of Armillaria root disease on future stand composition and structure which, for timber purposes, can be converted into volume losses. The latter is particularly significant since current expectations of timber yields over the next decade from certain forest areas in western North America may be overestimated by 50% because effects of Armillaria root disease have not been considered.

Even with the wealth of available empirical information, relationships that are appropriate for modeling disease behavior at the stand level need to be postulated. In modeling the dynamics and behavior of Armillaria root disease in forests of western North America, we found that the available information for specific components ranged from virtually no hard data to two or more conflicting data sets or opinions. Therefore, it became critical to document assumptions made during the modeling process because: (1) if model performance is questionable in certain areas, then the assumptions can be checked to see if they help to explain the concern; (2) if new information becomes available, then the current assumptions can be appropriately modified; and (3) if theory or concepts change in areas where little empirical information exists, then documentation of initial assumptions is necessary to consider any possible changes.

The model dynamically represents the spatial and temporal epidemiology of pathogenic *Armillaria* species or *P. weirii* (McNamee and others 1989, Stage and others 1990). It can project up to 40 growth cycles of stand development, normally of 10 years each, and operate in stands up to 100 ha. The three main components or submodels are root disease per se, "other agents," and an interface to a vegetation model (fig. 10.1). The root disease submodel provides the status and spread of

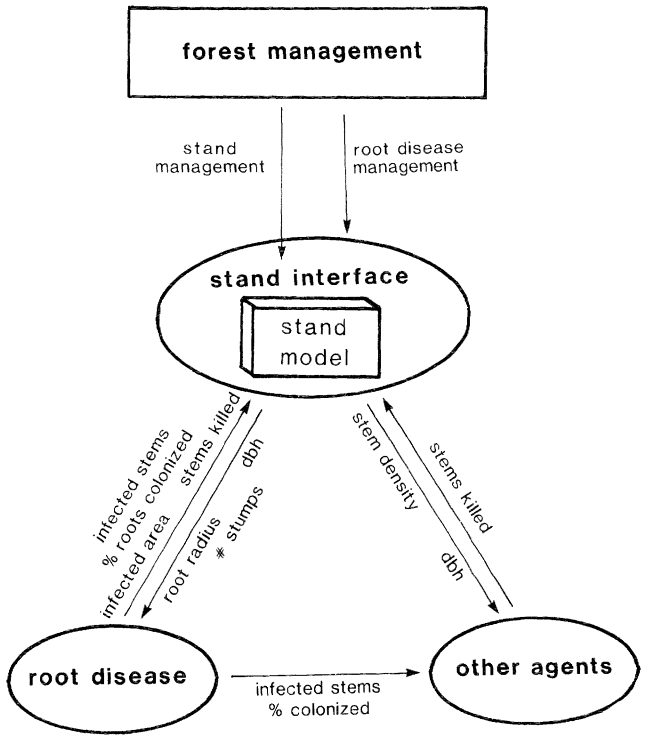


FIGURE 10.1. — Relationship among the three models of the Western Root Disease Model.

root disease and contains a Keyword mechanism to modify relationships to meet particular conditions (Stage and others 1990). This feature allows the user to explore alternative hypotheses concerning root disease dynamics. The "other agents" submodel simulates the effects of wind-throw and three types of bark beetle behavior. This submodel is important because it structures the interactions between root diseases and other mortality agents that can be important and damaging factors in forests of western North America (Shaw and Eav 1991). The stand-interface submodel links the stand-development model, to which the Western Root Disease Model must be attached, currently Prognosis (Stage 1973, Wykoff and others 1982), and the root disease and "other agents" submodels.

Critical Model Relationships and Associated Assumptions and Hypotheses

Spatial Resolution

The Western Root Disease Model spans two levels of organization: individual trees and the aggregation of these individuals into stands. Within a stand, two strata are defined with respect to root disease. The first consists of areas that are clearly beyond the influence of currently diseased trees. The second stratum consists of a number of root disease centers, each of which contains infected trees, uninfected trees, and other inoculum sources such as infected stumps. The size and separation of areas in these two strata define the spatial resolution of the model.

Within each stratum, the actual spatial proximity of individual trees is not maintained. When rates of pathogen spread to uninfected trees are calculated, however, the individual trees in a sub-sample of the first stratum are assigned x-y coordinates according to whether the stand is of natural origin (a random distribution is assumed) or is evenly spaced as in a plantation.

Center Dynamics

The model addresses three important characteristics of root disease centers: the dynamics of infection and inoculum within root disease centers; the expansion of root disease centers; and the carry-over of root disease to a new stand following stand entry.

Inside Established Centers

Progression Within Single Trees

The relationship that describes how live root systems become infected, trees are killed, and infection spreads in dead, infected roots (fig. 10.2) is a fundamental function of the model. This relationship was developed from the experiences and judgements of those who participated in model development. Chapters 4 and 5 provide background information relevant to these assumptions.

The relationship that describes the time between initial infection and death of a Douglas-fir tree on Douglas-fir habitat in the interior region of the Western United States is shown in fig. 10.3. This relationship is modified for other species and habitat types, but the hypothesis is that all trees react similarly to infection. The relationship represents, with some reference to published information (Hadfield and others 1986), the best professional judgement of the pathologists who participated in model development. They realized that, as modeled, the relationship may not be appropriate under situations of scattered mortality, a situation needing further research. For example, how is the percentage of a root system that is infected when a tree dies affected by *Armillaria* species, tree species, stress, etc? In recognition of these uncertainties, critical points of the relationship can be modified using the Keyword system (Stage and others 1990).

For example, one Keyword specifies the level of root infection at which trees die and allows users to vary the level for different tree species and sites. Another Keyword can be used to change the time-to-death for infected trees. A third Keyword allows users to modify infection and mortality dynamics by tree size.

A consensus of pathologists in western North America suggested the values in table 10.1 for the average portion of a root system that is colonized by *Armillaria* when a tree dies. Following tree death, the model

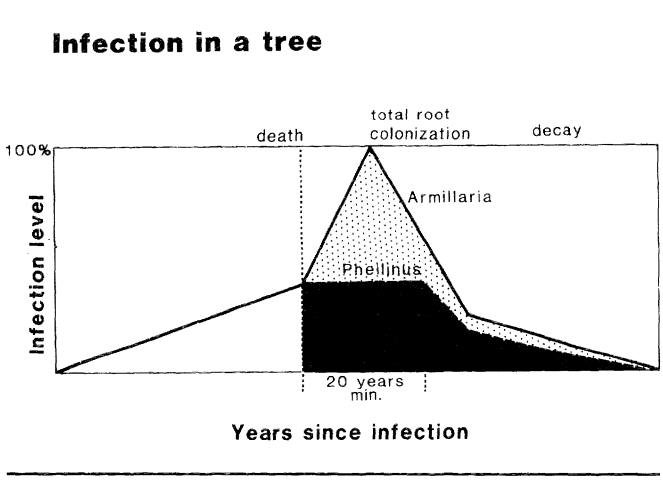


FIGURE 10.2 — Pattern of root pathogen spread and inoculum buildup and decline in a single tree root system.

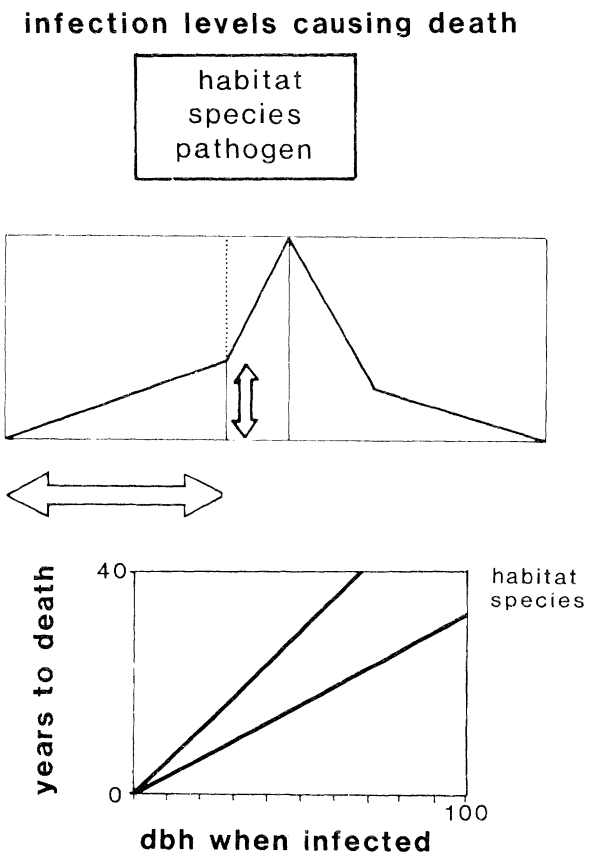


FIGURE 10.3 — Time required from infection by *Armillaria* to tree death for Douglas-fir on a Douglas-fir habitat type.

assumes that *Armillaria* colonizes all remaining portions of infected root systems within 5 years. Supporting evidence for this comes from Morrison (1981) and Shaw (1980).

The model assumes that dead trees or stumps can only become inoculum if, prior to their death or cutting, their root systems were already colonized to some degree with a pathogenic species of *Armillaria*. Even if a tree only has a small lateral lesion somewhere on its roots (Shaw 1980), its entire root system will, under this assumption, become inoculum within 5 years. Contrarily, trees not already infected at cutting, regardless of location, will not become inoculum. Even though this modeling assumption contrasts with certain hypotheses about the competitive saprophytic ability of *Armillaria* (see chapter 4, Garrett 1970), the model does provide sufficient inoculum for disease to progress in a manner judged to be reasonable by knowledgeable forest pathologists in western North America.

This assumption may be logical for modeling in coniferous forests of western North America. These forests show limited rhizomorph development by pathogenic species

TABLE 10.1 — Average percentage of root systems assumed to be infected at the time a tree is killed by root disease.

Tree species	Fungal Species	
	Armillaria	*P. weirii*
	(% root system infected)	
Douglas-fir	80	60
Pines	30	85
True fir	80	60
Hemlock	80	80
Spruce	75	65
Larch	00	75
W. redcedar	75	85

of *Armillaria* (Shaw 1980), and pathogenic lesions frequently occur on trees with little above-ground evidence of infection other than proximity to trees with obvious symptoms or signs of infection (see chapter 5). Perhaps the earlier assumptions on competitive saprophytic ability, developed primarily in the United Kingdom, need to be re-examined regarding current information on the pathogenicity of various *Armillaria* species and their relative in vivo abilities to produce rhizomorphs (see chapters 4 and 6).

In the model, how disease spreads through root systems of dead trees is independent of how the trees died, even though in reality the speed and mechanism of death may affect either the proportion of the root system actually colonized or the viability of resulting inoculum. This assumption relates to a fundamental research need regarding Armillaria root disease: the importance of, and mechanisms for, interaction of root diseases with other agents (both biotic and abiotic) of stress (see chapters 7 and 8; Shaw and Eav 1991).

The maximum lifespan of effective inoculum also may be affected by habitat type or other environmental parameters; however, users can modify these parameters. As modeled, the lifespan of effective inoculum is assumed to be a function of stump size and species, with rather rapid deterioration after maximum build-up (fig. 10.4). Species are grouped into heartwood (Douglas-fir, pines, and larch) and nonheartwood types (true firs, hemlocks, and spruce), with the latter decaying more rapidly. Inoculum is assumed to decay at a rate that reduces the radial extent of infected root systems by 75% during the first one-third of their lifespans. The remaining infected roots are assumed to decay at a steady rate over the remaining two-thirds of their lifespan. The pattern of inoculum decay is undoubtedly influenced by habitat type, tree rooting habit, temperature, moisture, and other abiotic factors not captured in the model but discussed in chapters 4 and 7.

Spread from Tree to Tree

Pathogen transmission to adjacent, living trees is modeled as a probabilistic process of two parts. First, an uninfested root system overlaps an infested system; and second, the pathogen will be transmitted given that the root systems overlap. The latter probability is controlled by a species-dependent Keyword. The first probability is calculated by simulation on a map which plots individual trees. Their spatial distribution is modeled as random (Poisson) for natural regeneration or a lattice for plantations, and can be changed by the user during the simulation. Kellas and others (1987) also suggest modeling tree distribution in Australian mixed species eucalypt stands with a Poisson distribution modified for stumps colonized by *Armillaria*.

Pathogen transmission via rhizomorphs is not explicitly modeled though one can change infection probabilities. Thus, increasing the probabilities of root system overlap, or pathogen transmission given root overlap, could be used to accommodate the activity of rhizomorphs where they are considered important agents of infection (see chapter 4).

Quantity of Inoculum

The quantity of inoculum available in a stand is estimated from the area occupied by infected roots. This area is determined by the relation of root extent to tree diameter and species, with rooting patterns assumed to be circular. If rhizomorph networks extend the influence of inoculum beyond the actual root systems boundaries (see chapter 4), then the areas could be increased. This action was considered to be unnecessary in forests of western North America because of limited in vivo rhizomorph production by the primary pathogenic species (see chapters 4 and 6). As described above, the size of these areas declines with time to represent the decay of root systems after tree death.

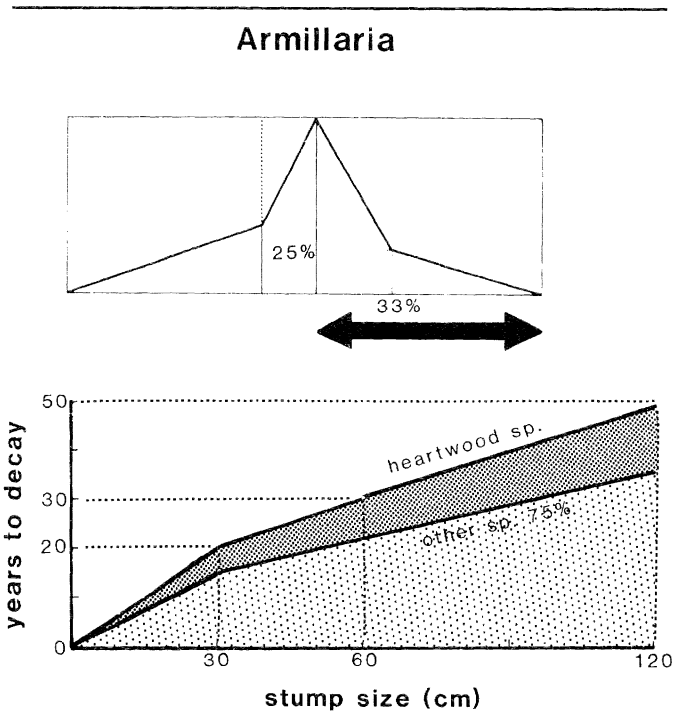


FIGURE 10.4 — Lifespan of *Armillaria* inoculum for trees with and without heartwood; see text for detailed description.

We know these assumptions have inherent inaccuracies and simplifications. For example, the density of roots is not uniform across a radius drawn outward from a stump, but decreases with distance from the stump. And, of course, trees, particularly when growing on slopes, do not have roots with a uniform, circular distribution. However, the important attribute is area, which is only slightly different for an ellipse than for a circle. A more detailed representation of actual root system geometry would have considerably complicated the model, with a limited likelihood of improving predictions.

Expansion of Centers

The simulation for enlarging infection centers has two main components: estimating the average rate of enlargement and translating that rate into a new stand area encompassed by root disease.

Rate of disease spread into uninfested area is simulated by a subroutine with enhanced spatial resolution. It maps a sample of the trees still outside the infection centers onto a smaller square area within which trees are assigned x-y coordinates. Then, the same relations that describe increases in area occupied by infected roots (fig. 10.2) and the probability of transmission, given that the root area of an uninfected tree overlaps that of a diseased tree, are used to estimate the time required for the disease to propagate across the width of the map. Width of map divided by the estimated time defines the radial spread rate of disease centers. The spread rate is thus not an input parameter but is calculated by the model. As such, it provides a means to gauge model performance as data on rates of infection center enlargement are available (see chapter 8). As an option, one can override this model function and input a static spread rate.

When this radial increment is added to the radius of each existing infection center, some centers may overlap. However, the new area of infection is calculated after adjusting for overlaps. The increase of infected area divided by the previously uninfected area pro-

vides a proportion for moving trees from the uninfected tree inventory to the infected tree inventory.

Carryover to Regeneration after Harvest

How root disease centers are affected by clearcutting and regeneration of a new stand is poorly understood. The modelers considered three different "carryover" scenarios: (1) root disease centers from the former stand cease to exist after clearcutting, and root disease in the new stand arises in a small number of new centers located within previously infected areas; (2) root disease centers from the former stand retain their integrity, and, as the new stand matures, these centers enlarge, starting at their old boundaries; and (3) after a clearcut and regeneration, root disease centers form around certain individual pieces of inoculum throughout the area affected in the former stand and these centers gradually expand and coalesce. These three scenarios actually form a continuum that depends on inoculum density and the probability of a piece of inoculum initiating a new center that is capable of expanding.

New disease centers have equal probability of occurring anywhere root disease occurred in the previous stand and no probability of occurring elsewhere. This assumption implies that all disease in the previous stand was noticeable and detected in the stand examination (see chapter 5) and that spores do not initiate new centers. We realize that the latter event must occur at some time (see chapter 9). However, for Armillaria root disease in western coniferous forests, in contrast to root disease caused by *H. annosum* in these same forests (Shaw and others 1989b), its occurrence seems to be infrequent enough that it can be ignored for stand-level modeling purposes—particularly when modeling stands that are already infected. Information presented in chapters 7, 8, and 9 supports this view.

A ring of root systems around the outside of each disease center represents trees that have just been infected. When these trees are cut, the prompt colonization of their entire root systems by the fungus causes disease centers to expand rapidly. In the model, the mean diameter of all root systems in the stand at the time of the cut is the distance by which radii of disease centers increase. Evidence for such action is found in Morrison (1981) and Shaw (1980). A major unknown is how far root disease actually does "jump out" from the recognized, above-ground edge of a center after clearcutting. Cursory examination of model behavior suggests that this is a sensitive parameter and thus it can be controlled by use of a Keyword. This feature of the model was most useful in preliminary work on adapting the Western Root Disease Model to represent root disease caused by *H. annosum* (Shaw and others 1989b).

Representation of Management Actions

Inoculum Removal

The Western Root Disease Model can simulate inoculum removal through "pushing" or removing infected stumps and their root systems (see chapter 11). This option can be requested in a specific year, with a factor specifying the efficacy with which roots are removed and the minimum diameter of dead trees and stumps to be removed. Even though this practice is an accepted management alternative in certain stands (Roth and others 1977), it is not universally applicable (Wargo and Shaw 1985).

Silvicultural Treatments

Regeneration systems ranging from single-tree selections to clearcutting can be simulated. Besides harvesting existing trees, new stands can be introduced following site preparation either by natural regeneration or by planting with species selected for disease resistance (see chapter 11). Likewise, particular species can be favored during thinning or during other partial stand harvests.

A full range of treatment alternatives may be considered when regenerating diseased stands, depending on economic constraints and stand management objectives. The most frequent approach to managing root disease problems in timber stands throughout western North America is regeneration to site-suited tree species that are disease tolerant (Hadfield and others 1986, Morrison 1981). The model can be used to compare the effects of various approaches. For example, the following options are among the many that may be compared and considered:

- No action—leave the stand "as is," but recognize presence of root disease.
- A clearcut, seed-tree cut, or shelterwood cut followed by natural regeneration. The mixture of species in the resulting regeneration will depend on the habitat type.
- Overstory removal to leave an understory of tree species that might be disease susceptible, disease tolerant, or a mixture of the two.
- A clearcut with stump removal followed by planting of a disease-susceptible but otherwise preferred species.
- A clearcut without stump removal followed by planting of a disease-susceptible but otherwise preferred species. Comparison to the preceding option provides an estimate of the control value of stump removal (see chapter 11).
- A base simulation, without invoking the Western Root Disease Model, of the Prognosis model for

stand development, perhaps followed by planting of a preferred but disease-susceptible tree species. These scenarios represent "control" simulations for the Western Root Disease Model.

Besides aiding management decisions, gaming with the model by preparing such scenarios can help scientists identify research needs relating to treatment alternatives. For example, the efficiency of stump removal (i.e., the proportion of inoculum removed) is a sensitive parameter in the model which suggests that carryover of root disease as a function of stump-removal efficiency is an important research area, particularly since little information exists on the topic.

What Data are Required?

The model is designed to start with sample inventories of actual stands. For example, the compartment examination procedure described by Stage and Alley (1972) and in the Forest Service Handbook for Region 1 (USDA Forest Service 1986) can supply the necessary stand data if it is augmented to include stumps infected with root disease (see chapter 5).

Besides the customary tree-size attributes, the model uses information on the frequency of tree infection by root pathogens. This value can be compiled by the model from disease status codes of the individual sample trees, or supplied by the user from an overall estimate based on an independent sample of the stand. The Western Root Disease Model also uses data on the area of the stand and the sizes and distribution of disease centers to initiate the simulation. The user may specify a total area in root disease and the number of centers. In this case, the model randomly locates root disease centers throughout the stand. Initially, each center will be of equal size, calculated as the total area in root disease divided by the number of centers. The alternative is to provide a list of root disease centers with X and Y coordinates and a radius for each center.

The model can start from bare ground by planting, by invoking the Regeneration Establishment component of the Prognosis model for stand development (Ferguson and others 1986), or from the stand description contained in the list of trees sampled in the inventory.

Conclusions

We believe that the Western Root Disease Model provides a workable framework for others to consider when modeling the behavior of Armillaria root disease in orchards or other forest situations. The current model should continue to improve as new information becomes available. The model is currently undergoing an analysis of its sensitivity to changes in the various parameters that control it and thus the assumptions and hypotheses under which it was developed (Marsden, unpubl.). We believe that the items to which the model is the most sensitive (longevity of inoculum and the quality, quantity, and type of input data) are the ones where additional resources could best be put to improve model performance. Thus, a list of research needs relevant to improving model reliability can be generated through a structured sensitivity analysis.

If the procedure we have outlined is used in model development, then it is critical that participants in the process represent a cross section of interested and knowledgeable scientists, managers, and administrators. Furthermore, it is paramount that scientists be willing to extrapolate beyond the limits of available data to help meet existing management needs. In so doing, however, they must insist that all extrapolations and assumptions are thoroughly documented. Also, such disease models need to be developed so that they can function in concert with existing models that may predict other stand or orchard attributes such as yield or watering regimes.

Based on how well users have accepted the Western Root Disease Model for both short-term, site-specific management decisions and long-range applications in planning, we strongly encourage others to pursue this avenue for transferring technology on Armillaria root disease dynamics into a useable tool for managers. We also contend that the process of doing so will help scientists clarify the current state of knowledge and help to focus management-oriented research needs.

CHAPTER 11

Avoiding and Reducing Losses from Armillaria Root Disease

Susan K. Hagle and Charles G. Shaw III

As the forest and agricultural land base is increasingly utilized, careful stewardship of remaining productivity becomes increasingly important. *Armillaria* epiphytotics can not only cause marked reductions in fruit and fiber production (see chapters 8 and 9), but they may also carry high economic, social, and ecological costs for control. In some forest settings, properly applied cultural control methods are efficient and effective. But for many crops, we lack convenient, cost-effective methods for control. In fact, it has been said (Schütt 1985) that while our biological knowledge about *Armillaria* has increased markedly since the time of Hartig, the efficiency of control measures, with some exceptions, has not improved very much. However, advances in our ability to identify species accurately (see chapters 1 and 2), determine their relative pathogenicities (see chapter 6), and model the disease process (see chapter 10) provide us a sharper image of disease problems, and should allow a more systematic evaluation of control options.

In this chapter, we examine various approaches and techniques for control and avoidance of Armillaria root disease in forests, orchards, and amenity plantings. These include use of resistant species, avoidance of hazardous sites, cultural manipulation, chemical application, biological methods, and integrated biological methods. Chapter 9 contains related material on management practices in plantations that can reduce losses from Armillaria root disease.

Armillaria species cause three types of disease in indigenous forests (see chapter 8). In one type, tree and shrub species are attacked and killed by an aggressive, primary pathogen (Filip 1977, Gibson 1960, Kile 1981, MacKenzie and Shaw 1977). In another type, the fungus lives primarily in chronic infections it causes on roots that may but seldom become aggressive. The third type causes butt rot that may or may not be related to other disease types (see chapters 5 and 6).

The first disease type often requires radical measures to effect control. The other two types may cause little damage if stand management maintains the resistance or tolerance of infected trees. In these latter two, a shift in the balance between the host and the pathogen induced by stresses such as drought, insect attack, other diseases, or anthropogenic activities can allow the fungus to expand and kill the host (see chapter 7).

The pathogenic behavior of *Armillaria* species in plantations, orchards, and amenity plantings also ranges from aggressive to benign. Control options may, however, differ from those that are feasible in indigenous forests, and they also may be more costly; however, the higher commodity values may offset higher costs (see chapter 9).

The type of root disease expression (see chapter 5) and the extent of damage are related to species and genotypes of *Armillaria* (Shaw and others 1981, Guillamin and Lung 1985, Rishbeth 1982, Kile and Watling 1988, Roll-Hansen 1985, Intini 1989a), inoculum characteristics (see chapter 4), inherent host resistance or tolerance (Thomas and Raphael 1935), host adaptation to site (Intini 1989a, Singh and Richardson 1973), stand structure and species composition, management history, and site factors which directly affect the pathogen (Redfern 1978, Blenis and others 1989; see chapter 6).

Where *Armillaria* acts as a secondary pathogen on plants that are predisposed in some way, control efforts should focus on the predisposing condition (see chapter 7). This problem becomes especially acute in situations such as those created by atmospheric deposition or photochemical oxidant injury, which are not only difficult to document but also difficult to control for societal reasons. In such cases, we could find ourselves treating symptoms at great expense with little benefit.

Where *Armillaria* is a primary pathogen, infection often leads to rapid death even if the plants were vigorous prior to attack. This distinction in pathogen behavior generally determines the type of control measure to use. Cultural controls often are used to reduce damage to natural stands or plantations of indigenous species,

while direct methods of inoculum removal, either alone or in combination with cultural control methods, may be required to reduce damage in plantations, arboreta, seed orchards, or amenity plantings.

Diseases can be avoided by using the natural balance and diversity of indigenous forests to prevent *Armillaria* epidemics, even though the fungus is present as a minor pest and natural thinning agent. Examples occur worldwide where *Armillaria* causes insignificant damage in indigenous forests but inflicts significant losses when these forests are cleared to establish exotic plantations (see chapters 8 and 9). The cost, both economic and ecological, of converting indigenous forests to exotic plantations must be weighed against any increased commodity value derived from the exotic species.

Needs Assessment

In many situations, *Armillaria* may be present in a forest or orchard and cause little damage. Thus, mere fungal presence is not sufficient cause to treat. Plants that are resistant to *Armillaria* throughout their lives or, as is the case with many conifers, through most of their lives, are capable of maintaining stand or orchard productivity. In such cases, *Armillaria* may act as a thinning agent in young stands (Filip and others 1989, Morrison 1981, Rishbeth 1972a) and as a nutrient recycler in old stands (Durrieu and others 1985, Mason and others 1989, see chapter 8). Disease often is severe in the first few years after plantation establishment, but subsides thereafter. Where this happens, primary inoculum from stumps or other buried woody material is the likely source of disease; secondary inoculum is not effective. Disease in New Zealand's radiata pine plantations (Roth and others 1979), in western North America's young ponderosa pine stands, whether planted or naturally regenerated (Hadfield and others 1986, Hagle and Goheen 1988, Morrison 1981), and in Europe's first-rotation conifer plantations on cleared hardwood sites (Hartig 1873b, Nechleba 1915, Pawsey 1973) follow this pattern.

A high incidence of mortality following establishment may be alarming, but without secondary spread of disease, the economic impact may be insufficient to justify control. Such is generally the case in indigenous ponderosa pine and coastal Douglas-fir stands of western North America (Morrison 1981, Hadfield and others 1986) and in many first-rotation conifers on former hardwood sites in Europe (see chapter 9). In contrast, radiata pine plantations on high-risk sites in New Zealand may lose 50% of the crop within the first 5 years after planting (van der Pas 1981b) which constitutes a severe impact. Direct reductions in primary inoculum (fig. 11.1) may be economically feasible in such

FIGURE 11.1 — Reduction of primary inoculum by removal of stumps and roots of indigenous forest cover in New Zealand prior to establishment of radiata pine plantations. Such actions can markedly reduce disease incidence and severity in first rotation crops (see fig. 9.7). (C. Shaw)

cases; even so, other alternatives also should be considered. For example, increased planting densities that allow for full stocking after suffering losses due to primary inoculum may, if effective, prove more economical and environmentally acceptable than efforts to reduce inoculum levels through stump removal or chemical treatment at the beginning of the rotation. Patchy killing of trees in the plantation may make thinning the remaining stand necessary after mortality has subsided. Contrarily, in orchards and amenity plantings, the economic importance of losing a few or perhaps even a single tree may be sufficient to justify inoculum removal or other costly control procedures. The lack of assessment data in these situations (see chapter 9) complicates decisions to implement control.

As indicated by Rishbeth's survey (1983) of gardens and forests in southern England, the species of *Armillaria* found on a stump, tree, or shrub can affect the decision to initiate control. For example, *A. gallica* had spread widely from an ash stump in a garden with no signs of attacking other trees or shrubs that it had encountered. However, *A. mellea* had spread from a *Prunus* stump and killed species of apple, stonefruit, birch, and sequoia. As identification of *Armillaria* species becomes more routine (see chapters 1 and 2), its use is likely to become standard before control is recommended.

Control Options

Silvicultural Considerations for Natural Forests

In natural forests, silvicultural control of Armillaria root disease is frequently an option. Local tree species grown in natural mixtures and densities may resist

Armillaria root disease even though they are known hosts for the local species of *Armillaria*. Where Armillaria root disease is a major concern in coniferous forests in western North America (see chapter 8), only indigenous tree species are grown in production forests. Even so, careful selection among species, seed sources, and cultural methods to match site conditions, particularly those related to habitat type (Daubenmire 1952) or site type (Corns and Annas 1986), is necessary to avoid economic losses. Using locally adapted seed sources for indigenous species that tolerate the disease is recommended for control in the Northwestern United States and Western Canada (Hadfield and others 1986, Morrison 1981, Williams and others 1989).

Substantial losses occurred in mixed coniferous forests of southern Oregon after selective harvest of ponderosa pine overstories (Filip 1977) caused a species composition shift to highly susceptible true firs. Severe root disease problems have been attributed to similar changes in species composition over much of the Western United States due both to selective logging of pines and larch and to fire suppression which favored shade tolerant true firs and Douglas-fir (fig. 11.2, see chapter 8). Dense Douglas-fir and true fir forests are unnatural on these sites and their development often results in disease conditions much like those found in exotic plantations. Current silvicultural practices in such areas, developed in part to reduce root disease losses, aim to re-establish pine, larch, and pine/larch mixtures with Douglas-fir and true firs composing less than 40% of the regeneration (Hagle and Goheen 1988).

Even when planted within their natural range, some species adapt poorly to certain sites. Although Douglas-fir is well distributed over diverse montane environments, the species has differentiated populations that are closely tied to elevation, latitude, and longitude (Monserud and Rehfeldt 1990, Rehfeldt 1982). Each population has adapted to local environmental conditions and fails to thrive when planted elsewhere. Other conifer species appear to behave similarly (Balmer and Williston 1983, Lotan and Perry 1983, Rehfeldt and others 1984). Thus, attention to seed sources is important for culturing these species. For example, substantial increases in *Armillaria* damage to Scots pine plantations in the German Democratic Republic followed a drought in 1969. Even after the drought, however, wildling pines were seldom affected by the pathogen, leading Kessler and Moser (1974) to recommend development of seed-saving methods to take advantage of natural resistance by regenerating stands through seeding with these sources.

Where use of locally adapted seed sources is not an option because the natural forests were removed, genetic differentiation within artificial populations can be used. Lung-Escarmant and Taris (1989) reported a method to test the *Armillaria* resistance of various pine species in natural stands. They suggested using the

FIGURE 11.2 — A natural forest in western Montana where ponderosa pine is more resistant to Armillaria root disease than most associated species. Although few large ponderosa pines remain in the overstory, past management practices that favored removal of ponderosa pine and excluded natural fire have allowed a Douglas-fir and true fir understory to develop that is more susceptible to Armillaria root disease. (S. Hagle)

method to test Maritime pine for population, family, and clonal differentiation in resisting *A. ostoyae* within the pine's natural range in southwest France. Whether considering indigenous or exotic trees, genetic differentiation should be matched to the natural site conditions where the trees are growing. Intraspecific variation in adaptation to sites may be great, but the extension of a species' range may still be limited. For example, dieback and declines of silver fir plantations in central and northern Italy are frequently associated with infection by *A. ostoyae*. The diseases appear to be drought-triggered and to be concentrated in fir plantations established in an area "phytoclimatically inferior and warmer" than natural sites for the species (Intini 1989a).

Excessive moisture may have been responsible for the demise of several ponderosa pine plantations which were established during the 1940's in Idaho using nonlocal seed sources (Hagle unpubl.). The parent trees, growing more than 500 km away, were on very different sites than those where the plantations were established. The plantations were installed to determine if genotypes adapted to dry pine sites would produce superior growth when planted on more mesic, grand fir climax sites. The trees grew exceptionally well for about 40 years but died rapidly thereafter from a combination of pests, among which Armillaria root disease was most prominent (Hagle unpubl.). Ponderosa pine has since been found to have "seed zones" of limited range (Squillace and Silen 1962) and planting outside these zones is not recommended. McDonald (1990) discusses how the potential for ecophysiological maladaptation of species to specific sites may influence their susceptibility to Armillaria root disease.

Avoiding Hazardous Sites

Matching indigenous species with suitable sites is one way to minimize disease hazard. Sites can be hazardous because they predispose the host in some way, as with off-site plantings. Sites with heavy inoculum loads of pathogenic *Armillaria* species may also be hazardous. Whether a naturally high frequency of *Armillaria* infections occurred in the previous stand or human or other activity increased the level of infection, the influence of inoculum loading is little disputed (see chapter 4). Hazardous sites also may result from site conditions that are unusually favorable to disease development; however, these conditions are difficult to discern because of our limited knowledge of *Armillaria* ecology. Site hazard varies within indigenous forests. For example, soil-related differences in disease severity were reported in Norway spruce stands in central Europe (Gramss 1983). Enhanced survival of stands was partially attributed to the "poor podzolic highland soil types" to which the spruce appeared to be better suited. The highland soils distinguish low-hazard sites for growing Norway spruce. Ono (1970) found edaphic, topographic, and vegetational relationships with the level of *Armillaria* damage to Japanese larch plantations in Hokkaido, Japan. Williams and Marsden (1982) related root disease patch occurrence in coniferous forests in Montana and Idaho to certain productive soil and habitat types. Byler and others (1990) found that the more productive habitat types in these areas had greater root disease severity than the less productive types, a result that was partially supported in a preliminary report by McDonald and others (1987a).

Damage in exotic plantations or orchards can be minimized by establishing them on sites with a low disease hazard. Sokolov (1964) reported soil types in the Soviet Union influenced the severity of Armillaria root disease in mulberry plantations. Severe Armillaria root disease in Norway spruce stands in Poland was related to a combination of soil type and elevation (Mańka 1980). The spruce plantations were established on sites previously supporting indigenous stands of silver fir and common beech. Whitney (1984) found Armillaria root disease to be more severe on conifers in Ontario, Canada, where soils were coarse-textured and sandy rather than finer-textured and silty.

Vegetation on a site prior to clearing for establishing an orchard or plantation may indicate differences in root disease hazard. For example, Leach (1939) reported site hazard differences for tea plantations associated with indigenous stands of Muula trees in eastern Africa. Muula roots remain alive but moribund for years after cutting, and these were most often associated with root disease in tea plantations. In Kenya and New Zealand, differences in root disease severity also were noted on lands converted to radiata pine from different indigenous forest types (Gibson 1960, Shaw and Calderon 1977).

Hendrickson (1925) reported Armillaria root disease in fruit orchards to be of "widespread economic importance in California." He considered oak roots from the indigenous forest to be the most important source of inoculum, but secondary spread among orchard trees maintained the disease long after the land was cleared of oaks. Cooley (1943) surveyed Eastern U. S. fruit orchards and found little disease except in the sandhill section of North Carolina. Most orchards in this area had been established on land cleared of hardwood forests, indicating that a heavy load of primary inoculum that harbored a pathogenic species of *Armillaria* may have been responsible for the frequency of disease. Orchards themselves may maintain a high hazard from one rotation to the next. For example, *Armillaria* inocu-

lum from a highly susceptible cherry rootstock created hazardous site conditions in replanted orchards of several species in Michigan (Proffer and others 1987).

Vegetation maintained on a site from one crop to the next may affect disease hazard. Although conifer plantations in Britain are damaged by *Armillaria* in the first 10-15 years after planting on sites freshly converted from hardwoods, succeeding rotations of conifers sustain much less damage (Pawsey 1973). Balsam fir Christmas tree plantations are damaged in the first few years after establishment on sites converted from indigenous mixed hardwood and pine forests (Wargo and Houston 1987). Similarly, radiata pine plantations in New Zealand may be severely damaged within 5 years after conversion from indigenous forest but subsequent rotations of pine on the sites may be little damaged (Shaw and Calderon 1977). If this effect is found to be consistent, such areas would be high-hazard sites only for the first rotation after conversion. Recent work in New Zealand, however, suggests that the cursory observations on limited disease incidence in second- or third-rotation pine crops by Shaw and Calderon (1977) may have been premature (MacKenzie and Self 1988).

Resistance

In situ host resistance to root disease is a complex topic as it involves the genetics of both host and pathogen as well as environmental influences. It also can involve managing mixtures of genotypes with varying levels of resistance. Some species with superior resistance or tolerance to infection in one location may be quite susceptible in other locations. For example, Douglas-fir is among the species recommended for planting in Britain where Armillaria root disease is especially damaging to Scots pine plantations (Greig and Strouts 1983). It is also considered more resistant than Sitka spruce and grand fir in France (Delatour and Guillaumin 1985). Within its natural range in western North America, however, Douglas-fir is considered rather susceptible to Armillaria root disease (Hadfield and others 1986, Hagle and Goheen 1988). In all three locations, *A. ostoyae* is the most common parasite of conifers (Rishbeth 1982, Guillaumin and Lung 1985, Morrison and others 1985a).

Even within limited geographic areas, such discrepancies in resistance can be seen. For example, ponderosa pine's resistence is superior to true firs and Douglas-fir over most of western North America (Morrison 1981, Hadfield and others 1986), but natural ponderosa pine stands in some south-central Washington, sites are severely damaged by Armillaria root disease (Shaw and others 1976a, Shaw and Roth 1976). Douglas-fir associated with pine on these sites suffers less damage (Roth and Rolph 1978). A similar situation exists in the Jemez Mountains of northern New Mexico (Wood 1982, Shaw unpubl.). Thus, intraspecific variation in adaptation to sites may be as great as interspecific variation within the natural ranges of any two or more species.

Radiata pine planted in New Zealand suffers considerable damage from Armillaria root disease (Shaw and Calderon 1977), but the same species is not particularly damaged in its natural range in western North America (Raabe 1979a). This variation is probably related to differences in *Armillaria* species, inoculum loads, or edaphic, climatic, or physical site characteristics. Whether endemic or exotic, genetic differentiation in tree species should be compared to site conditions and pathogen species in natural settings in which plants are to be grown, and plantations should be monitored for suitability of the genotypes to the site. Diseases and insect attack are likely to be among the earliest indications of poor compatibility of trees with growing sites.

Relative resistance of many species has been observed in forests, orchards, and parks as well as by controlled inoculation experiments (see chapter 6). Much of this work was completed before many of the pathogenic *Armillaria* species were recognized. As such, the information is only useful in a general way. Morquer and Touvet (1972b) noted considerable variation in resistance of several conifers, but found no species immune to infection by *Armillaria*. Pines were notably susceptible while certain ecotypes of Norway spruce were relatively resistant. Mugala and others (1989) found that white spruce succumbed more readily than lodgepole pine in potted-seedling studies, but these results were inconsistent with field observations where white spruce was damaged less than lodgepole pine (Blenis and others 1987, 1989). In Kenya, Gibson (1960b) noted that slash pine was more severely affected than either radiata or Mexican weeping pine. Rishbeth (1972a) reported that, in a 17-year-old mixed stand of Scots pine and Norway spruce, large patches of pine died while the spruce was virtually unaffected. Day (1927b) observed that, in adjacent 3-year-old plantations of Scots pine and Japanese larch, the pines were more frequently infected than larch but relatively fewer pines were killed. Up to 10% of the larch were killed by *Armillaria*.

Resistant rootstocks have been developed for both fruit and fiber species (Raabe 1966a). Rhoads (1948) reported considerable variation in susceptibility of citrus rootstocks to *Armillaria* and Thomas and others (1948) tested rootstocks of prune and apricot for resistance in California. Armitage and Barnes (1968) reported that loblolly pine resisted *Armillaria*, and a heterospecific graft of slash pine onto loblolly rootstock was sufficiently resistant to replace slash pine killed by the fungus. Peach, almond, apricot, and cherry trees are severely damaged by *A. mellea* in France, while plum is generally resistant

(Guillaumin and others 1989b). The use of resistant rootstocks appears to be the only practicable control option in French stone fruit orchards (Guillaumin and others 1989b). Two rootstocks in particular, resulting from interspecific crosses between diploid plum and peach, appear to satisfy both *Armillaria* resistance and other cultural demands. Heaton and Dullahide (1989b) recommended a number of *Armillaria*-resistant plum, pear, and grape rootstocks for Granitebelt orchards in southern Queensland, Australia.

Perhaps gene manipulation techniques can improve a species' adaptability and physiological resistance to disease, produce populations or clones immune to *Armillaria*, or improve economic qualities of endemic, resistant species (Hubbes 1987). Rootstocks for fruit orchards are prime candidates for receiving resistance genes. Superior quality rootstocks which produce desired growth and compatibility characteristics could be made resistant to *Armillaria* if genes known to produce successful resistance reactions in other species or genotypes can be identified and transferred to the genome of the otherwise superior rootstock. Research to determine the relative resistance of different species and genotypes under a variety of conditions must continue. Additional information is also needed on the nature of resistance (i.e., physiological, genetic, or environmental) and its interaction with various *Armillaria* species and genotypes (see chapter 6).

Using species resistant to *Armillaria* may be economically practicable in some cases, and their suitability may be enhanced by combination with other control procedures. Planting mixtures of species with differing resistance to *Armillaria* may reduce secondary spread of disease in a plantation. For example, Morrison and others (1988) observed fewer and smaller disease patches in plots planted with highly susceptible lodgepole pine or Douglas-fir in alternating rows with resistant western redcedar or paper birch, compared to plots planted only to lodgepole pine or Douglas-fir. Presumably the benefit was derived from limiting secondary disease spread, which limited the size of infection centers.

Other Cultural Considerations

Regeneration methods may influence tree condition and susceptibility to Armillaria root disease. For example, Newfoundland conifer plantations that originated from bareroot stock were significantly more damaged by *Armillaria* than those that had been broadcast seeded (Singh and Richardson 1973). Similar results were reported for Scots pine plantations in the German Democratic Republic (Kessler and Moser 1974). Seeding is not always acceptable, however, because of other advantages that accrue from planting (Page 1970, Schubert and others 1970).

Manipulation of rotation length may minimize losses in some situations. As previously mentioned, Norway spruce may be only moderately affected by Armillaria root disease. Still, in Czechoslovakian plantations, diameter growth significantly decreased in 70- to 80-year-old spruces (Hřib and others 1983). The current recommendation is to harvest stands by this age, which represents the culmination of mean annual increment. Such pathological rotations are used primarily for economic reasons. They often are an option in hardwood forests in England (Greig and Strouts 1983) and the Eastern United States (Marquis and Johnson 1989), and conifer stands in Ontario, Canada (Whitney 1988b). Stands or individual trees are harvested prior to the age at which they are expected to succumb to Armillaria root disease (Marquis and Johnson 1989). A similar principle underlies a recommendation to use training and trellising techniques to promote early cropping in Australian fruit tree orchards where secondary spread of *Armillaria* causes high losses (Heaton and Dullahide 1989b). This practice allows an early recapture of investments and minimizes the economic impact caused by eventual loss of trees to Armillaria root disease.

Both precommercial and commercial thinning to reduce damage from *Armillaria* should be considered according to the type of disease caused. Where host condition has little influence over infection and killing by *Armillaria*, thinning is unlikely to reduce losses and may increase damage by providing additional food bases. Precommercial and commercial thinning to reduce intertree competition may effectively reduce disease losses where *Armillaria* is a secondary pathogen, as it is in hardwood forests in southern England and the Eastern United States. Suppressed oaks and pines (completely shaded by overtopping trees) were extensively infected by *A. mellea* and *A. ostoyae* in southern England. Subdominant (codominant) trees in those stands were only slightly infected and apparently resisted extension of infection by these pathogens (Davidson and Rishbeth 1988). Red spruce stands in the Northeastern United States have been severely damaged by Armillaria root disease following stagnation from overstocking (Wargo and Shaw 1985). Early thinning has avoided such problems in most managed stands. Precommercial thinning of conifer stands in western North America has produced mixed results (Hagle and Goheen 1988). In one ponderosa pine plantation, mortality rates in thinned and unthinned plots were similar 20 years after precommercial thinning (Filip and others 1989); however, net productivity on the thinned plots increased because of superior volume growth. Singh (1981a) recommended precommercial thinning in pine and spruce stands in eastern Canada to reduce stress and improve resistance to *Armillaria*.

Morrison (1981) advised delaying precommercial thinning in western Canada to 30 years in infection centers of conifer stands where losing residual trees after early thinning could cause low stocking. Blenis and others (1987) observed infections in young lodgepole pine stands in Alberta, Canada, and noted that rhizomorphs spread from the previous stand's debris. They concluded that precommercial thinning could be done after such debris was no longer effective as inoculum.

Interpreting how Armillaria root disease responds to commercial thinning or other forms of partial harvest is difficult because stands are often affected by mixtures of two or more root pathogens capable of varying responses. However, in ponderosa pine stands infected with *Armillaria* alone, a partial harvest increased mortality even though the total area affected had remained unchanged (Shaw and others 1976a). Rishbeth (1978b) reported indirect evidence of *A. mellea* spores colonizing oak and ash thinning stumps and thereby causing a low incidence of new infection patches. On this basis, he recommended delayed thinning. Such a delay might, however, lead to even larger and more numerous new infection patches through spore infection of even larger stumps. Severe damage has occurred in New Zealand kiwifruit orchards following spore infection of stumps created by removing windbreak trees (Horner 1988).

Kellas and others (1987) surveyed for *A. luteobubalina* infection in mixed eucalypt stands in Victoria, Australia, where shelterwood cutting has become common. Cutting intensity did not appear to influence the incidence of disease in residual trees and stumps, but frequent, low-intensity cuts may have increased infection. The high infection frequency on residuals in thinned stands also may have reduced growth and increased mortality sufficiently to negate any growth response in the thinned stands.

Partial harvest for commercial thinning or other purposes has caused considerable damage to conifer stands in the Western United States. Byler and others (1986) reported that root disease frequency doubled in stands that had at least one harvest entry compared to those with no tree cutting. Losses to root diseases have been so great following partial harvests which leave stands composed primarily of Douglas-fir or true firs (fig. 11.2) that these silvicultural methods are not recommended on sites prone to root disease in the Western United States (Hadfield and others 1986, Williams and others 1989). Such management activities have truly exacerbated the incidence and severity of Armillaria root disease.

Soil pH, organic content, and nutrient status are partially alterable in forestry and orchard operations. Very little direct evidence, however, establishes the effects of fertilization or other soil amendments on Armillaria root disease in forest crops. This may be, in part, because the nutritional requirements of forest species are not well understood. Rykowski (1981a) was not able to decrease mortality rates by applying fertilizers in young Scots pine plantations, but the appearance of chronically infected trees did improve. Fertilization increased the fungistatic effects of extracts from periderm and phloem tissues but also improved the nutritional quality of wood used during the saprophytic phase of *Armillaria* colonization (Rykowski 1983). Application of potassium did, however, significantly reduce damage from *Armillaria* in banana plantations in Malawi (Spurling and Spurling 1975). Use of fertilizers as a management tool in orchards and plantations is further discussed in chapter 9.

Direct Reduction of Inoculum

On the speculation that buried roots and stumps were a source of infection in young orchard trees, Barss (1913) suggested that stumps and roots be removed and that non-orchard species be cropped for several years before establishing an orchard. Since then, recommendations for physical removal of inoculum (Shaw and Roth 1978, 1980) have involved removing diseased trees, uprooting stumps (Roth and others 1980, Arnold 1981), destroying stumps and root remnants (Morrison and others 1988), and turning the earth over to a considerable depth (Heaton and Dullahide 1989b, Horne 1914, McGillivray 1946, Reitsma 1932, Sokolov 1964).

The quantity and location of inoculum that must be removed to prevent disease buildup and spread and the cost of removal justified by the crop's future value are difficult to balance. Complete eradication of the fungus by mechanical, biological, or chemical means is improbable (Williams and others 1989) and of doubtful value. Even sites that have been devoid of woody material for decades, such as land supporting an herbaceous crop, may be re-invaded, albeit slowly, through spore infection when placed into woody plant production (Rishbeth 1978b). Soil disturbance may also stimulate fresh rhizomorph production, increasing the disease risk to newly planted trees (Morrison 1976, Redfern 1970). Excessive removal of woody debris from a site may be detrimental to mycorrhizae as well (Harvey and others 1981, Maser and others 1984). Such additional risk must, however, be evaluated in context with the large quantities of inoculum removed.

Stump and root removal is commonly practiced in preparing sites for fruit orchards and other high-value crops, whether converting from indigenous forest or removing infected trees from the previous crop. This procedure has long been standard where *Armillaria* is a

recognized threat (Wallace 1935, Thomas and Raphael 1935, Hendrickson 1925). In forests, Sokolov (1964) recommended inoculum removal in the USSR, and Morrison and others (1988) successfully reduced inoculum on an infested site in western Canada. The latter treatment involved pushing the trees, roots and all, from the ground followed by a raking which removed most roots over 1.5 cm in diameter. Douglas-fir, lodgepole pine, western redcedar, and paper birch were planted on treated and untreated sites. After 20 years, mortality of Douglas-fir and lodgepole pine in untreated plots was 5-10 times greater than in treated plots.

Areas within a managed, natural ponderosa pine forest in south-central Washington were so severely damaged by *Armillaria* that they had little likelihood of persisting as a commercial forest unless the disease was controlled (Shaw and others 1976a). Here, recently killed sapling and pole-sized trees, and those positioned where they were likely to be infected (Shaw 1980, Shaw and Roth 1974), were pushed over with a bulldozer (fig. 11.3). This method removed nearly intact root systems. Removing infected pine trees and stumps during thinning in this forest has also effectively reduced disease losses (Roth and others 1977, Roth and Rolph 1978). Special guidelines (fig. 11.4) are used to mark symptomatic trees before pushing them over (Roth and others 1977, 1980). Extraction of stumps up to 50 cm in diameter with a vibratory stump puller has also been successful in this area (Arnold 1981).

In New Zealand, Armillaria root disease increased the cost of growing first-rotation radiata pine on sites with a high disease hazard by approximately 40% (Shaw and Calderon 1977). This cost approximates the maximum amount available for disease control. Initial stump uprooting and removal trials (fig. 11.1) suggested that the high disease losses encountered on nontreated sites could be reduced at a cost that was within the calculated economic limits (Shaw and Calderon 1977, Shaw unpubl., van der Pas 1981b). Utilizing stumps and roots may partially offset removal costs (Arnold 1981, Hakkila 1974). In addition, stump removal and plowing can benefit general seedling performance (Department of Forestry Queensland 1972, Morrison and others 1988, Roth and others 1977, Sorochkin 1972).

FIGURE 11.3 — Pushing trees over with a bulldozer, rather than cutting them off with a saw, is an effective way to dislodge root systems and remove inoculum on certain soil types. The technique may also be used to create a "root free zone" that may serve as a barrier to further disease spread (see fig 11.4). (C. Shaw)

The value of plants in urban forests, gardens, and orchards often justifies the much higher treatment costs that are associated with inoculum removal. Recommended site preparation for orchards in the Australian Granitebelt consists of ripping to 35 cm so large roots can be removed, then ripping again to 25 cm to remove roots as fine as 1 cm (0.4-inch) diameter, followed by hand removing remaining roots. Without such rigorous site preparation, *A. luteobubalina* kills up to 50% of the newly planted fruit trees (Heaton and Dullahide 1989b). In New Zealand, soil has been extensively sifted to remove even the smallest infected root pieces (fig. 11.5A) to rehabilitate highly susceptible kiwifruit orchards that have suffered severely from *Armillaria* (Horner 1988). In urban settings, Pawsey (1973) recommended removing stumps and roots to whatever degree is practicable.

Stump and root removal is most effective in the following situations. First, removal works well where the pathogen causes a primary disease from existing inoculum but does not continue to spread from secondary inoculum. Second, if secondary inoculum is important in the disease process, then it occurs in distinct patches (not diffuse in stands) and thus makes careful removal over concentrated areas possible. Third, where secondary inoculum is important but the crop can be managed on a short rotation, such as intensively managed fruit orchards, the pathogen has little time for secondary spread from unremoved inoculum pieces.

Stump removal followed by a brief fallow period may increase the effectiveness of inoculum reduction where secondary inoculum is of concern. The presumably small, residual roots would likely decay quickly—particularly in tropical regions. Pits dug for removing infected coffee roots in Kenya either are left open for several months or are treated with a soil sterilant before covering. Still, a short fallow of 3-4 months after treatment is recommended (Baker 1972). Extended fallow periods without stump removal might also prove effective in temperate regions although costly in terms of lost production time (Roth and others 1977). Rishbeth (1972b) showed that hardwood stumps cut 40

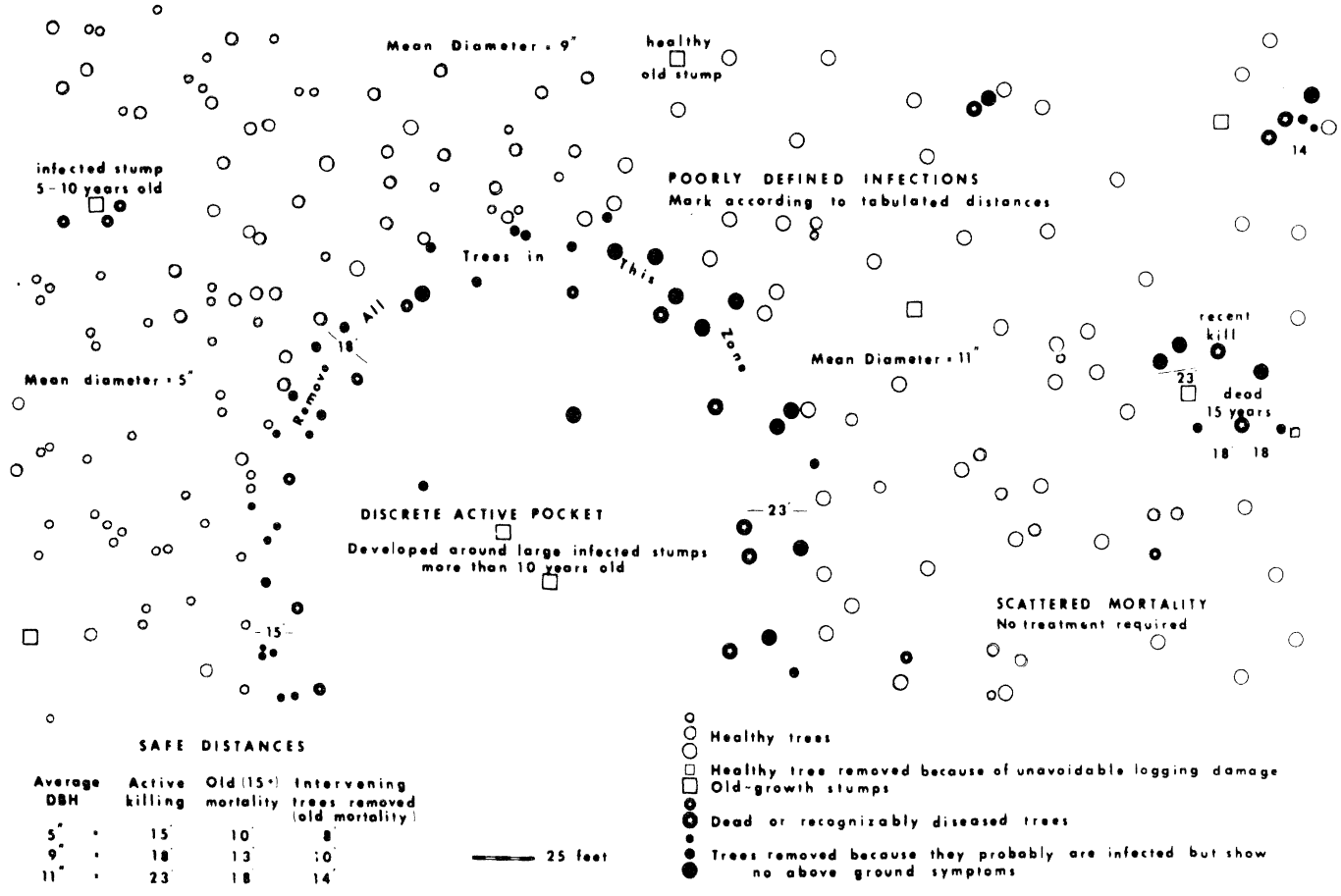


FIGURE 11.4 — Guidelines developed by Roth and others (1977) to treat Armillaria root disease in ponderosa pine in south-central Washington. Trees at the left of the *Armillaria* center are too small for commercial thinning and are not disturbed except for removals to establish a barrier at the limits of the disease pockets. This action is done in conjunction with treatment of the commercial stand at the right by pushing trees over to remove their root systems (see fig. 11.3). (L.F. Roth, Journal of Forestry)

years earlier produced rhizomorphs, but at much lower levels than from younger stumps. The disease is rarely troublesome in first-rotation forests established on natural grassland and former agricultural land. However, the economics of extending the rotation by delaying establishment may prohibit leaving land unstocked for lengthy periods—unless the land can otherwise be profitably used (e.g., livestock grazing or herbaceous cropping) in the interval. Annual cropping with cereals or alfalfa 4-5 years following clearing of orchard sites may deplete most of the nutritional sources for *Armillaria* (Guillamin 1977, Mallet and others 1985) while providing income.

Simply avoiding old planting sites when replanting sections of orchards killed by *Armillaria* is advised for many crops (Horner 1990, Baker 1972, Heaton and Dullahide 1989b). This, combined with stump and root removal, provides an effect similar to fallow before root closure occurs. Heaton and Dullahide (1989b) recommended avoiding specific locations from which infected trees have recently been removed in orchards by relocating new orchard rows midway between the former rows.

Trenching at least 1.1 m deep to isolate infected plants from healthy parts of a vinyard or fruit orchard is used in France to control spread of Armillaria root disease (Guillamin 1977). Trenching 0.6 m (20 inches) in cocoa plantations (Rishbeth 1980) and 1.1 m (3.5 feet) deep in coffee plantations (Wallace 1935) has been used successfully in Africa. Laying a plastic barrier in a trench and then backfilling it with removed soil has also been used for disease control in kiwifruit orchards in New Zealand (fig. 11.5B).

Pruning and girdling diseased roots (Kendall 1931), or drying and aerating the root collar (Munnecke and others 1976, Kendall 1931), may be useful on individual, high-value trees in orchards, parks, gardens, forest

A

B

FIGURE 11.5 — Control of Armillaria root disease in kiwifruit orchards in New Zealand (see fig. 9.6). A: Sifting of soil to remove even small pieces of infected roots to rehabilitate a severely diseased orchard. This method is no longer cost effective due to a reduction in kiwifruit profitibility. (I.J. Horner) B: If properly placed, trenches lined with plastic and backfilled serve as a mechanical barrier to root and rhizomorph growth into yet-unaffected portions of the orchard. (R.A. Hill)

campsites, and seed orchards (Shaw and Roth 1978, 1980). Levitt (1947) used a water jet to expose root collars of infected citrus trees. Rackham and others (1966) reported that exposing root collars effectively controlled *Armillaria* in citrus orchards, but they also noted that the large craters formed by this control method over a number of years could pose a hazard to workers in the orchards. Sokolov (1964) reported control of *Armillaria* in young Siberian larch by exposing the root collar. Generally, these procedures would be inappropriate in commercial forests because of prohibitive costs.

Chemical Protectants, Eradicants, and Curatives

Apart from soil fumigation with carbon disulphide, methyl bromide, or chloropicrin after removing woody debris, very little experimental evidence supports the effectiveness of the most commonly advocated chemical treatments (Shaw and Roth 1978). Justification for using many of these treatments is based on superficial and subjective criteria (Pawsey and Rahman 1976a). Reviews of chemical control of *Armillaria* have been presented by Pawsey and Rahman (1976a), Shaw and Roth (1978, 1980), and Thies and Russell (1984). As emphasized by Shaw and Roth (1978 1980), managers must understand whether chemical applications are intended to protect uninfected plants, eradicate the fungus in infected stumps and roots, or treat or cure infected, living plants.

Chemical soil fumigants that destroy *Armillaria* in root fragments are especially useful in orchard, vineyard, and floriculture operations where agricultural methods are applicable (Kissler and others 1973). Methyl bromide, a chemical demonstrated useful for this purpose in 1935 (Richardson and Johnson 1935), is still the most extensively used fumigant because of its non-specific action and good penetrability in soil (Vanachter 1979). Activity of chloropicrin against *Armillaria* in prune root sections was demonstrated in 1936 (Godfrey 1936). It is still a much-used fumigant because it will destroy even the most resistant soil pathogens, although penetration in soil is difficult to achieve. Fruit crops in California have benefited from using carbon disulphide injected at regular intervals over an infected site after removing stumps (Bliss 1951). Heaton and Dullahide (1989b) also recommended using methyl bromide fumigation in orchards by injection into root-free soil and sealing with plastic.

Systemic fungicides which have effectively suppressed *A. ostoyae*, *A. mellea*, and *A. gallica* in vitro are hexaconazole, flutriafol, and fenpropidin (Turner and Fox 1988). Chemicals of the ergosterol biosynthesis inhibitor type are promising candidates for protectants and curatives (Schwabe and others 1984). In fact, systemic fungicides, "which can act both directly on the fungus in the soil and within the plants at some distance from the point of application, have raised great hopes for the control of soil-borne fungal diseases"

(Louvet 1979). However, as Louvet (1979) also points out, substantial problems need to be solved before application of systemic chemicals is successful. First, translocating systemics in plants is acropetal whereas basipetal translocation would be more useful in treating roots. Second, strains of pathogens resistant to their action may rapidly appear in crops although evidence for such action in *Armillaria* is currently lacking.

Armillatox, a phenolic emulsion containing 48% active ingredients (unidentified), has been marketed for specific use against *Armillaria*. Apparently, the compound was developed after successfully controlling *Armillaria* with creosote (Bray 1970). Pawsey (1973), however, considered creosote to be phytotoxic, and of doubtful value. Penetration of the material into the wood is minimal. Armillatox did produce some phytotoxic effects at the recommended dilution, even though rhizomorph production was somewhat reduced (Redfern 1971, Pawsey and Rahman 1976b). There was no evidence of remedial effect of Armillatox on established root infections. Pawsey and Rahman (1974) suggest that repeated, regular use of Armillatox might protect against rhizomorph-initiated infection. Redfern (1971) found no beneficial effect from the chemical.

Maneb (Pawsey and Rahman 1976a) and boric acid (Heško 1971, Pawsey and Rahman 1976a) applied to tree root collars and stumps have successfully reduced some rhizomorph production, although Shaw (unpubl.) abandoned trials with boric acid because of severe phytotoxicity. Rykowski (1974b) suggested that field applications of sodium pentachlorophenate (NaPCP) protected young Scots pine from *Armillaria* infection, helped eradicate the fungus in infected stump roots, and did not injure the tree. However, Shaw and others (1980) found NaPCP did not reduce infection on radiata pine inoculated with *Armillaria*, but they did notice some decreases in host vigor. The long-term benefit of such treatments has yet to be demonstrated, and considerable doubt remains about the phytotoxic effects of both boric acid and NaPCP (Shaw and Roth 1978), and about NaPCP's potential effects on human health (Shaw and others 1980).

Filip and Roth (1987) applied chemical to the root collars of small-diameter ponderosa pines to prevent mortality caused by *A. ostoyae* in south-central Washington. After 10 years, none of the seven chemicals (benomyl, captan, copper sulfate, iron sulfate, copper wire, vorlex, or chloropicrin) appeared to reduce mortality. Although single applications of the chemicals to protect pines from lethal infections were not effective, some of the chemicals may protect pines in high-value areas, such as seed orchards, recreation sites, or ornamental plantings, where economics may justify repeated applications.

Fedorov and Bobko (1989) tested several fungicides for controlling existing infections in live hosts by applying them to the rhizosphere. They reduced rhizomorph production using cuprozan, fundazol, derozal, topsin-M, and copper oxychloride, but doubted the overall benefit of the treatments because *Armillaria* remained alive in host tissues. Recently, treating stone fruit trees in Australian orchards with potassium phosphite (fig. 11.6) has shown promising results; 75% of the treated trees appear to be recovering from *Armillaria* infection (Heaton and Dullahide 1989a).

In many chemical tests, effects on rhizomorph production have been the main criterion for effective treatment. However, Redfern (1975) reported a significant negative correlation between the percentage of trees killed by *Armillaria* isolates and dry weight of rhizomorphs produced by the isolate. Rishbeth (1985a) also reported greater rhizomorph production by weakly parasitic *A. gallica* compared to the more aggressive *A. mellea* and *A. ostoyae*. Conceivably, treatment could alter the stump or rhizosphere environment such that the resident *Armillaria* species change, which may result in a difference in rhizomorph abundance. Considering our current understanding of rhizomorph production among different species in situ, this criterion for evaluating treatment effectiveness should be reconsidered (see chapter 4).

Filip and Roth (1977) successfully controlled *Armillaria* in ponderosa pine stumps using methyl bromide, Vorlex, chloropicrin, carbon disulphide, and Vapam (fig. 11.7). Chloropicrin, Vortex, and methyl bromide eliminated the fungus from the stumps. In high-value crops and ornamentals where stump removal may not

FIGURE 11.6 — Injection of a peach tree with potassium phosphite as treatment for prior infection by Armillaria root disease. Development of epicormic branches along the stem indicates success. Infected trees can apparently recover following such treatments, but resumption of full production remains to be shown. (J.B. Heaton)

A B C

FIGURE 11.7 — Chemical treatment of ponderosa pine stumps to eradicate *Armillaria*. Holes are drilled in stumps (A) as entry ports for liquid eradicant chemicals (B) or gaseous fumigants (C). Many such treatments successfully eliminated *Armillaria*, but costs were considered too high for general applications in forestry. (Filip 1976, Filip and Roth 1977). (G. Filip)

be desirable or possible (such as where access with heavy machinery needed for removal is limited), fumigants may be a useful option. Fumigant injections to establish barriers which prevent vegetative spread of *Armillaria* may also be valuable (Houston 1975, Filip and Roth 1977) in forestry applications.

The significant economic losses caused by *Armillaria* justify further efforts in chemical control (Pawsey and Rahman 1976a). In certain situations, chemical treatments may alter disease development at the epiphytotic level (Filip and Roth 1977). However, certain aspects of such work need to be stressed. For example, field studies must define treatments by specific objectives—i.e., protecting, eradicating, or curing. Disease condition prior to treatment (i.e., proportion of stump colonized) must be known. Techniques for assessing effectiveness must be both valid and definitive. The cost/benefit of treatment must be evaluated in context with alternative measures and crop value. Detrimental effects of the treatment on the environment or society require consideration. One potential advantage for chemical protectants is that the critical region for application is likely to be the root collar, thus limiting the area requiring treatment. For seedlings, a protectant chemical should be relatively inexpensive, safe, easy to handle, easy to apply at planting, nonphytotoxic, fungitoxic or fungistatic, and persistent in the region of application. As discussed below, the control achieved by some chemicals may be interrelated with their effects on other microorganisms.

Biological Control and its Integration with Other Methods

Biological control of a plant pathogen has several inherent advantages (Hunt and others 1971). Among others, it is more likely to be accepted by the public than either chemical control or the expense and initial unsightliness of stump and root removal. To control *Armillaria*, a rhizosphere or wood-inhabiting organism might function by inhibiting or preventing rhizomorph and mycelial development, by limiting the pathogen to substrate already occupied, by actively preempting the substrate, or by eliminating *Armillaria* (perhaps through replacement) from substrate already occupied. Pursuing these potential benefits must, however, be tempered with the feasibility of the technique (Shaw and Roth 1978, 1980).

Rishbeth (1976) noted two important features that make control of *Armillaria* by introduced organisms difficult. First, *Armillaria* has a positional advantage over introduced fungi since it already may occupy a portion of the substrate. Second, although *Armillaria* does not colonize wood quickly, it spreads rapidly in

the cambial zone of freshly killed trees. He suggested that antagonistic organisms might not be able to prevent *Armillaria* from becoming established in stumps, but they may restrict further stump colonization and thus limit the available food base. The same logic has been used to suggest that *Armillaria* species of limited pathogenicity may serve as biological control agents for *Heterobasidion annosum* (Fr.) Bref. (Morrison and Johnson 1978; Shaw 1989b,c).

Perhaps the most thoroughly studied antagonists of *Armillaria* are *Trichoderma* species (fig. 11.8) from which two fungitoxic substances, trichodermin and an unidentified compound, have been isolated (Ishikawa and others 1976). Aytoun (1953) studied in vitro interactions of *Trichoderma* and *Armillaria* and concluded that *Trichoderma* must be considered a possible controlling factor in the spread of pathogenic fungi. Sokolov (1964) found fungi in six genera, including *Trichoderma*, *Penicillium*, and *Peniophora*, antagonized *Armillaria*. He recommended using *T. viride* Pers.:Fr. as a control for Armillaria root disease. Dubos and others (1978) found that the medium in which *T. viride* inoculum was grown altered the degree to which the antagonist inhibited rhizomorph production by *Armillaria*. Morquer and Touvet (1972a) suggested growing *T. viride* for *Armillaria* control on a "lactoserum" medium.

Trichoderma species are common and ubiquitous soil inhabitants (Aytoun 1953, Griffin 1972), which might suggest that applying *Trichoderma* inoculum is generally unnecessary. *Trichoderma* has been implicated in *Armillaria* control using sublethal doses of fumigants (Bliss 1951, Ohr and others 1973, Filip and Roth 1977),

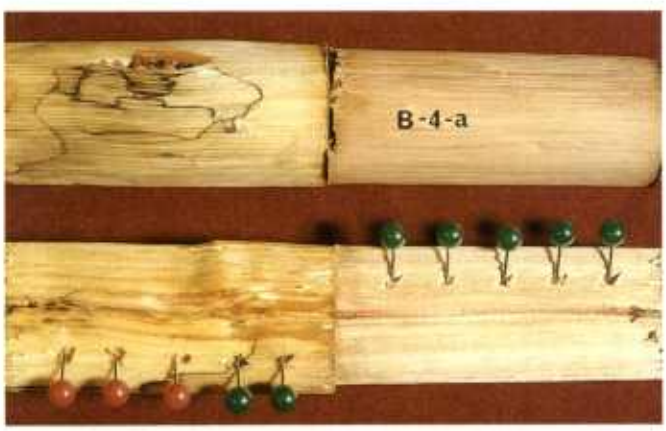


FIGURE 11.8 — Test of antagonism between *A. luteobubalina* and *Trichoderma* sp. Karri wood blocks previously colonized by *A. luteobubalina* (left) and *Trichoderma* sp. (right) were placed face-to-face in soil for 6 weeks, separated and split, and isolations made at 1-cm intervals back from the contacting faces of each block. Green pins indicate recovery of *Trichoderma* sp., red pins *A. luteobubalina*. Penetration of *Trichoderma* sp. into the wood block previously colonized by *A. luteobubalina* was apparently stalled by the zone line. (E. Nelson)

sublethal heating or drying treatments (Rackham and others 1966, Munnecke and others 1976), and possibly fire (Reaves and others 1990). *Scytalidium lignicola* Pesante also produces a toxin with antifungal properties toward *Armillaria* (Cusson and LaChance 1974). *Armillaria* growth in culture is halted by either *Scytalidium* or its toxin, scytalidin. Since both *Trichoderma* and *Scytalidium* are common in soil, the basis for improving their ability to control *Armillaria* lies in shifting the balance among the fungal populations. An inability to maintain effective populations of organisms antagonistic to *Armillaria* under field conditions has been the main factor limiting successful biological control (Shaw and Roth 1978, 1980).

Bliss (1951) demonstrated the ability of *T. viride* to replace *Armillaria* in artificially infected root segments fumigated with carbon disulphide (CS_2). Garrett (1957, 1958) showed that CS_2 can directly damage *Armillaria* mycelium; pure cultures of *T. viride*, in the absence of fumigation, also killed *Armillaria*. Apparently, both direct fumigant toxicity and subsequent action of *T. viride* were killing *Armillaria* in fumigated soils. After fumigation (fig 11.7C), Filip and Roth (1977) frequently isolated *T. viride* from pine stumps in which *Armillaria* was no longer viable. Munnecke and others (1973) suggested that after fumigation with CS_2 or methyl bromide, a lag period for *Armillaria* growth occurred, indicating a "weakening" of the *Armillaria*. *Trichoderma viride*, being more tolerant of the chemical (Ohr and others 1973), was able to exploit the lag period and exert an antagonistic action on *Armillaria*.

Riffle (1973) noted that two mycophagous nematodes greatly reduced mortality of ponderosa pine seedlings inoculated with *Armillaria*. The nematodes apparently reduced fungal vigor and growth. In vitro studies of how mycophagous nematodes affect mycelia of *Armillaria* and *Trichoderma* species indicated a possible role of *Aphelenchus avenae* in controlling *Armillaria* in a French vineyard. The nematode destroyed the hyphae of *Armillaria* but grew well on *T. polysporum* (Link ex Pers.) Rifai without reducing its growth (Cayrol and others 1978).

Armillaria produces antibiotic compounds (see chapter 3). Oduro and others (1976) suggested that such activity may be an important factor in surviving attack by antagonistic soil microorganisms. Significantly, Ohr and Munnecke (1974) showed that sublethal methyl bromide fumigation prevented the production of antibiotics by *Armillaria*. Munnecke and others (1976) suggested that heating or drying may similarly affect *Armillaria*. The critical factor is that *Armillaria* is stressed. The factors causing the stress may concurrently stimulate antagonistic organisms, resulting in further damage to the already weakened *Armillaria*.

Direct competition for the woody substrate may be an important natural control of *Armillaria*. Garrett (1956b) hypothesized that root-inhabiting parasites would have a low competitive saprophytic ability (see chapter 4). Redfern (1968) suggested that *Armillaria* probably cannot survive indefinitely as a saprophyte and that control is perhaps most easily achieved in the saprophytic phase. Leach (1937) observed that *Rhizoctonia lamellifera* Small prevented *Armillaria* from colonizing tea roots. Sokolov (1964) also observed that spruce stumps colonized by *Lenzites saepiaria* Fr. and *Peniophora gigantea* (Fr.) Massee were not invaded by *Armillaria*. From laboratory tests, Orlos (1957) thought *Fomes pinicola* (Swartz:Fr.) Cke. might be useful in controlling *Armillaria* because of its greater growth rate and ability to exclude *Armillaria* from occupied media. Fedorov and Bobko (1989) tested 10 basidiomycetes which were capable of excluding *Armillaria* from occupied substrates. Two of these, *Peniophora gigantea* (Fr.) Massee and *Pleurotus ostreatus* (Jacq.:Fr.) P. Kumm., also effectively prevented *Armillaria* growth in freshly cut stumps into which they had been inoculated.

Coriolus versicolor (L.:Fr.) Quél, *Stereum hirsutum* (Willd.:Fr.) S.F. Gray, and *Xylaria hypoxylon* (L.:Fr.) Grev. inoculated into karri thinning stumps simultaneously with *A. luteobubalina* (fig 11.9) each significantly reduced colonization by *Armillaria* (Pearce and Malajczuk 1990b). The eucalypt stumps were colonized both above and below ground by the competing fungi, but they were more effective antagonists above ground. A naturally occurring, cord-forming species of *Hypholoma* proved to be even more competitive with *Armillaria*, in some cases excluding it entirely.

Such cord-forming, wood-decay fungi have a similar niche to *Armillaria* and are perhaps the most exciting recent discovery in relation to its possible biological control. They are capable of subcortical mycelial growth in stumps and occupy the same initial sites as

A

B

FIGURE 11.9 — Successful establishment of antagonistic fungi in karri stumps inoculated with *A. luteobubalina*. Stump inoculation with either *Coriolus versicolor* (A) or a *Hypholoma* sp. (B) significantly reduced colonization by *A. luteobubalina*. The competing fungi colonized the eucalypt stumps both above and below ground, but were more effective competitors above ground. A naturally occurring, cord-forming species of *Hypholoma* proved to be even more competitive with *Armillaria*, in some cases excluding it entirely (Pearce and Malajczuk 1990b). (E. Nelson)

Armillaria (Redfern 1968). According to Rayner (1977), cord formers "closely paralleled *A. mellea* in their behavior, except for their lack of pathogenicity." Further studies have indicated that several species of cord-forming basidiomycetes, in particular *Phanerochaete velutina* (DC per Pers.:Fr.) Karst., *Hypholoma fasciculare* (Huds.:Fr.) Kumm., and *Steccherinum fimbriatum* (Pers.:Fr.) J. Erikss., have considerable potential to spread and colonize woody debris in field sites. Several produce networks of mycelial cords in soil and litter (Dowson and others 1988a) which can infest additional woody substrates (Dowson and others 1988b). Populations of some cord formers can be manipulated by chemically treating stumps (Rayner 1977), a finding that indicates potential to artificially induce biological control of *Armillaria*. Ammonium sulphamate appears particularly useful as it increased colonization and decay by cord-formers of below-ground portions of treated beech and birch stumps (Rayner 1977).

Stump fumigation has excluded or eradicated *Armillaria* directly in lethal doses (Bliss 1951, Rackham and others 1966, Filip and Roth 1977). Sublethal doses, however, do not kill *Armillaria* directly, but allow competing fungi less affected by the chemical to replace it. (Munnecke and others 1973, Ohr and others 1973). Sublethal doses of methyl bromide injected into orchard soil in California successfully controlled *Armillaria* (Munnecke and others 1981). *Trichoderma* spp., which resisted the methyl bromide, may have been responsible for controlling *Armillaria* in the fumigated soil. Formaldehyde was used in sublethal doses to control *Armillaria* in apple and pear orchards in China (Chang and others 1983), where *Trichoderma* populations were stimulated by the treatment and were credited with the control.

Silvicides which can rapidly kill host tissues are used to kill trees before cutting in tropical regions where the killed roots decay rapidly and pathogens are readily replaced by saprophytes (Mallet and others 1985). However, in temperate regions, the rapid killing by herbicides may benefit *Armillaria*. For example, Rishbeth (1976) reported that stump treatment with 2,4,5-T favored *Armillaria* colonization; and Pronos and Patton (1979) found more rhizomorph production 10 years after treatment in stumps of herbicide-killed oaks than in girdled oaks. Rapid death from the herbicide treatment was thought to have favored *Armillaria* over competing saprophytes.

While stump colonization is an important factor in Armillaria root disease, the nutritional quality of stumps and roots will influence the longevity of the pathogen. Leach's report (1937) that ring-barking effectively controlled the spread of *Armillaria* in African tea plantations led to several investigations into the nutritional suitability of altered stumps for *Armillaria*. Leach (1939) suggested that decreased concentrations of stored carbohydrates within the roots of ring-barked trees rendered them unsuitable for *Armillaria* colonization. Ring-barking also could have rendered the roots more easily colonized by other, saprophytic fungi and thus quickly reduced the volume of material available for *Armillaria*.

Redfern (1968) found that ring-barking or poisoning mature oaks 1 year prior to felling in Britain resulted in more rapid decay of the roots by *Armillaria* compared to those felled without prior treatment. Rhizomorph production may have been a major influence in Redfern's study. While rhizomorphs may be scarce in African soils (Wiehe 1952), they are abundant and proliferate epiphytically on live tree roots in Britain where localized lesions on live roots are also common. Ring-barking and silvicide treatment favored the invasion of the already present parasite. The roots of the treated oaks either initially were not a good substrate, or deteriorated quickly because significantly fewer rhizomorphs were produced from sections of treated roots compared to roots of non-treated trees 5 years after felling. Neither ring-barking nor silvicide treatment was effective in reducing mortality in subsequent plantations.

Lanier (1971) indicated that girdling old Scots pine and common beech a year before felling reduced the number of young pines attacked. Disease incidence was low, however, even in untreated parts of the forest. Punter (1963) indicated that girdling reduced neither the mortality of young trees nor the number of *Armillaria* basidiomes on stumps. Swift (1970) concluded that ring-barking effectively prevented invasion of stumps from external inoculum sources, but spread of the fungus from pre-existing lesions was not inhibited and probably was enhanced.

Heating and drying methods such as those employed by Birmingham and Stokes (1921) and Rackham and others (1966) are costly, difficult to apply, and almost certainly limited in utility to orchard and ornamental situations. Broadcast burning after clearfelling of indigenous forest in New Zealand significantly reduced the number of viable rhizomorphs compared to counts before clearfelling (Hood and Sandberg 1989). However, all the pine plantations described in New Zealand as having suffered severe losses from *Armillaria* (Shaw and Calderon 1977) were on sites burned during preparation for planting. Apparently, such reductions in rhizomorphs are not sufficient to control the disease. Focan and others (1950) noticed that root disease increased among perennial plants established after burning, and *Trichoderma* spp. markedly declined. However, Reaves and others (1990) reported that recovery of

Trichoderma isolates from soil was unaffected by fire. The species composition shifted such that, after burning, the most frequently isolated species had a greater antagonism towards *Armillaria*.

Mycorrhizae have been suggested as a protection against parasitic attack by *Armillaria*. Gaudray (1973) postulated that the formation of mycorrhizae on exotic Sitka spruce in France is incomplete and thus affords inadequate protection against *Armillaria*. Studies in vitro have shown that mycorrhizal fungi can inhibit *Armillaria* (Eghbaltalab and others 1975). Direct protection by mycorrhizae seems unlikely, however, as the main infection sites for *Armillaria* are on coarse roots rather than the fine roots where mycorrhizae develop.

Examining natural populations of organisms that are antagonistic to the identified, parasitic species of *Armillaria* present on sites that express different severities of root disease may be useful. Such examinations may indicate characteristics that could be manipulated through management so as to favor antagonistic organisms.

Conclusions

Whether to invest in *Armillaria* control and selecting control methods are decisions that need to be based on the value of losses in the absence of control. Assessing impact to commodity production or other features such as amenity value may, in itself, be costly, but it is an important precursor to any control decision. Both monetary and environmental costs of various control alternatives should be justified by commodity or other values derived. Effectiveness of control in a particular situation is another important consideration. For example, stump removal that reduces inoculum by 60% may greatly improve the productivity of a crop for which primary inoculum is the major concern, or of a crop that is produced in a short time. But stump removal with this same level of inoculum reduction may only slightly improve the productivity of a crop subject to secondary disease spread.

Control projects must be monitored long-term in most crops to measure relative gains from treatment (Jančařík 1955). Fruit orchards that are subject to secondary spread of *Armillaria* may require monitoring over decades to evaluate treatment fully. Forests may require a century or more to reach maturity. If disease spread from secondary inoculum is of concern, then monitoring for at least 20-30 years may be required to assess control effectiveness. How the effectiveness may carry over into subsequent rotations also needs to be considered because little information is available on this subject (see chapter 10). In such cases, interim evaluation and some degree of faith in projections are necessary.

In summary, the following checklist needs to be considered before any attempts are made to control Armillaria root disease.

(1) Critically evaluate disease impact to ensure that the level of loss justifies control. The use of disease models may aid this effort (see chapter 10).

(2) Control through cultural modifications should be given first priority, particularly in forests. As our current forest management rarely emulates nature's processes, pathologists must work in direct cooperation with foresters to understand and modify disease-stimulating practices.

(3) Utilize resistant or tolerant species, genotypes, or rootstocks, if known, that are compatible with other necessary values. Ensure that the host genotype selected for resistance is suitable for planting on potential sites, and will provide for the planned end use of the fruit or fiber. Pursue opportunities to genetically engineer *Armillaria*-resistant or tolerant species.

(4) When establishing new plantations or orchards, exercise care in site selection. Small-scale trials to evaluate disease potential should be established prior to large-scale land clearing and plantation or orchard development. If the site is found to have a high disease hazard, then one must be prepared for costly preestablishment actions such as inoculum removals by more thorough site preparation, postponement of plantation or orchard establishment for some unknown period, or elimination of the site from further consideration.

(5) Maintain the general health of the forest, orchard, or amenity planting by preventing damage from other agents, avoiding adverse sites, and discouraging detrimental human activities.

(6) Direct reductions of inoculum levels by physical removal of stumps and roots requires careful economic and ecological analysis. The effectiveness of such treatments in orchards, exotic plantations, and amenity plantings is generally appreciated; their effectiveness in natural forests will be better understood within the next few years when results from existing, long-term trials become available.

(7) When considering chemical treatments, clearly differentiate among protectants, eradicants, and

curatives. Except for high-value fruit or amenity trees, curatives are likely to be uneconomical. Even in orchards and amenity plantings, chemical applications need to be realistically evaluated for their relative cost/benefit. For chemical treatment of stumps, consider compounds that can be translocated, particularly basipetally. Protectants should be inexpensive, easy to handle and apply, nonphytotoxic, fungitoxic or fungistatic, and relatively persistent. Possible environmental and human health hazards require consideration.

(8) Fumigation, girdling, and silvicide treatment before felling may be useful methods to employ in preparing land for orchards, ornamentals, and some forestry applications such as seed orchards and test plantations. Fallowing after such treatment may improve effectiveness, especially where disease spread from secondary inoculum is anticipated.

(9) Biological control is desirable but requires further development for practical application in most situations. Research on antagonists, particularly cord-formers, needs to continue as does work on the various actions (i.e., fire, chemicals) that might be used to alter conditions in a way that favors developing and maintaining populations of desirable, antagonistic organisms.

Armillaria control needs to be a thoughtful, reasoned process, not a random or haphazard one. Evaluating the necessity for control and, if found necessary, determining the best option to implement are integral to prudent stewardship of forests, orchards, and amenity plantings. Our increased understanding of species identity, their pathogenic behaviors, and ecological relationships offers the opportunity for a systematic evaluation of approaches to controlling Armillaria root disease. Combined with results from long-term tests of inoculum removal and evidence of some new developments in genetic resistance, chemical effectiveness, and biological methods, success should be greater in the future than it has been in the past.

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Scientific and Common Names of Plants Noted in This Book

Common Names — Scientific Names

COMMON NAMES	SCIENTIFIC NAMES
acacia	*Acacia*
African oilpalm	*Elaeis guineensis*
Agathis	*Agathis*
ailanthus	*Ailanthus*
albizia	*Albizzia falcata* Merr.
albizia	*Albizzia*
alder	*Alnus*
almond	*Prunus amygdalus* Batsch.
alpine ash	*Eucalyptus delegatensis* R.T. Bak.
American chestnut	*Castanea dentata* (Marsh.) Borkh.
American beech	*Fagus grandifolia* Ehrh.
apple, pome fruit	*Malus*
apricot	*Prunus armeniaca* L.
araucaria	*Araucaria*
Arizona pine	*Pinus arizonica* Engelm.
Arizona pine	*Pinus ponderosa* var. *arizonica* (Engelm.) Shaw
ash	*Fraxinus*
avocado	*Persea*
Bahaman pine	*Pinus caribaea* var. *bahamensis* Barr. ex Golf.
balsam poplar	*Populus balsamifera* L.
balsam fir	*Abies balsamea* (L.) Miller
banana	*Musa*
banksia	*Banksia*
barkclothtree	*Brachystegia*
beech	*Fagus*
beefwood	*Casuarina*
beet	*Beta*
bigtooth aspen	*Populus grandidentata* Michx.
birch	*Betula*
black currant	*Ribes nigrum* L.
black oak	*Quercus velutina* Lamarck
black spruce	*Picea mariana* (Mill.) B.S.P.
blackberry, raspberry	*Rubus*
broad-leaved peppermint	*Eucalyptus dives* Schau.
broom	*Cytisus*
brown barrel	*Eucalyptus fastigata* Deane & Maid.
brown salwood	*Acacia mangium* Willd.
cacao	*Theobroma cacao* L.
cactus	*Opuntia*
cane	*Arundinaria*
Caribbean pine	*Pinus caribaea* Morelet
carrot	*Daucus*
cassava	*Manihot*
ceanothus	*Ceanothus*
cedar	*Cedrus*
cedrela	*Cedrela*
chaulmoogratree	*Hydnocarpus*
cherry	*Prunus*
chestnut	*Castanea*
chocolatetree	*Theobroma*
cinchona	*Cinchona*
citrus	*Citrus*
cocoa	*Theobroma*
coconut	*Cocos*
coffee	*Coffea*
colanut	*Cola*
common ash	*Fraxinus excelsior* L.
common fig	*Ficus carica* L.
common teak	*Tectona grandis* L. f.
common chaulmoogratree	*Hydnocarpus anthelminticus* Pierre
common beech	*Fagus sylvatica* L.
common pistachio	*Pistacia vers* L.
common pomegranite	*Punica granatum* L.
common tea	*Camellia sinensis* (L.) Ktze.
cork oak	*Quercus suber* L.
Corsican pine	*Pinus nigra* var. *maritima* (Ait.) Melville
cotton	*Gossypium*
cryptomeria	*Cryptomeria*
currant, gooseberry	*Ribes*
cypress	*Cupressus*
cypress pine	*Widdringtonia*
cypress pine	*Callitris*
dacrydium	*Dacrydium*

COMMON NAMES	SCIENTIFIC NAMES
dawn redwood	*Metasequoia*
deodar	*Cedrus deodora* G. Don ex Loud.
Douglas-fir	*Pseudotsuga menziesii* (Mirb.) Franco
Douglas-fir	*Pseudotsuga*
downy oak	*Quercus pubescens* Willd.
eastern white pine	*Pinus strobus* L.
eastern hemlock	*Tsuga canadensis* (L.) Carr.
elm	*Ulmus*
Engelmann spruce	*Picea engelmanni* Parry ex Engelm.
English oak	*Quercus robur* L. (Q. *pendunculata* Ehrh).
eucalypt-gum	*Eucalyptus*
European larch	*Larix decidua* Mill.
falsecypress	*Chamaecyparis*
fig	*Ficus*
fir, true fir	*Abies*
fish pelargonium	*Pelargonium hortorum* Bailey
flooded gum	*Eucalyptus grandis* Hill ex Maid.
geranium	*Pelargonium*
gliricidia	*Gliricidia*
gmelina	*Gmelina*
gmelina	*Gmelina arborea* L.
gooseberry, current	*Ribes*
granadilla	*Passiflora*
grand fir	*Abies grandis* (Dougl. ex D. Don) Lindl.
grape	*Vitis*
greatcone banksia	*Banksia grandis* Willd.
green wattle	*Acacia decurrens* (Wendl.) Willd.
grevillea	*Grevillea*
guava	*Psidium*
Hankow willow	*Salix matsudana* Koidz.
hazelnut	*Corylus*
hemlock	*Tsuga*
hickory	*Carya*
hinoki	*Chamaecyparis obtusa* Endl.
Honduran pine	*Pinus caribaea* var. *hondurensis* Barr. & Golf.
Honduras mahogany	*Swietenia macrophylla* King
hops	*Humulus*
Hungarian oak	*Quercus frainetto* Ten.
incense-cedar	*Calocedrus decurrens* (Torr.) Florin (*Libocedrus decurrens* Torr.)
incense-cedar	*Calocedrus*
Indian fig	*Opuntia ficus-indica* Mill.
Indian pipe	*Monotropa hypopitys* L.
Indian pipe	*Monotropa*
Indian pipe	*Monotropa uniflora* L.
irontree	*Metrosideros*
jack pine	*Pinus banksiana* Lamb.
Japanese redcedar	*Cryptomeria japonica* (L.) D. Don
Japanese larch	*Larix leptolepis* (Sieb. & Zucc.) Gord.
jarrah	*Eucalyptus marginata* Donn ex Smith
jujube	*Zizyphus*
karri	*Eucalyptus diversicolor* F. Muell.
kauri	*Agathis australis* Salisb.
Khasi pine	*Pinus kesiya* Boyle ex Gordon (*P. insularis* Endl.)
khaya	*Khaya*
kiwifruit	*Actinidia*
Korean pine	*Pinus koraiensis* Sieb. & Zucc.
larch	*Larix*
lavender	*Lavandula*
leadtree	*Leucaena*
lime	*Citrus aurantifolia* (Christmann in L.) Swingle
litchi	*Litchi*
loblolly pine	*Pinus taeda* L.
locust	*Robinia*
lodgepole pine	*Pinus contorta* Dougl. ex Loud.
loganberry	*Rubus loganobaccus* L. Bailey
loquat	*Libotrya*
macadamia	*Macadamia*
Mahaleb cherry	*Prunus mahaleb* L.
mahogany	*Swietenia*
mango	*Mangifera*
maple	*Acer*
Maritime pine	*Pinus pinaster* Ait.
messmate stringybark	*Eucalyptus obliqua* L'Hérit.
Mexican weeping pine	*Pinus patula* Schiede & Deppe
Mexican cypress	*Cupressus lusitanica* Mill.
mlanji cedar	*Widdringtonia whytei* Rendle
Morinda spruce	*Picea morinda* Link
mountain pine	*Pinus uncinata* Mill. ex Mirb.
mountain ash	*Eucalyptus regnans* F. Muell.
mountain gray gum	*Eucalyptus cypellocarpa* L. Johnson
mountain hemlock	*Tsuga mertensiana* (Bong.) Carr.
mulberry	*Morus*
Muula	*Parinari mobola* F. Muell. ex Benth.
myrtle-beech	*Nothofagus cunninghamii* (Hook. f.) Oerst.
narrow-leaved peppermint	*Eucalyptus radiata* Sieb. ex DC
New Guinea gum	*Eucalyptus deglupta* Blume
nightshade	*Solanum*
northern California walnut	*Juglans hindsii* Jeps. ex Smith
Norway spruce	*Picea abies* (L.) Karst.
oak	*Quercus*
ohia	*Metrosideros polymorpha* (Gaug.) Rock

COMMON NAMES	SCIENTIFIC NAMES
oilpalm	*Elaeis*
olive	*Olea*
orchid	*Gastrodia cunninghamii* Hook. f.
orchid	*Gastrodia elata* Bl.
orchid	*Gastrodia*
orchid	*Galeola*
orchid	*Galeola septentrionalis* Reichb. f.
papaya	*Carica*
paper birch	*Betula papyrifera* Marsh.
paraserianthes	*Paraserianthes*
paraserianthes	*Paraserianthes falcataria* (L.) I. Nielsen
parsnip	*Pastinaca*
passion fruit	*Passiflora*
pawpaw	*Asimina*
peach	*Prunus persica* Sieb. & Zucc.
pear, pome fruit	*Pyrus*
pecan	*Carya illinoiensis* (Wangenh.) K. Koch
Persian walnut	*Juglans regia* L.
persimmon	*Diospyros*
pindrow fir	*Abies pindrow* Royle
pine	*Pinus*
pistachio	*Pistacia*
planetree	*Acer pseudoplatanus*
pomegranite	*Punica*
ponderosa pine	*Pinus ponderosa* Dougl. ex Laws.
poplar	*Populus*
potato	*Solanum tuberosum* L.
provence broom	*Cytisus purgans* (L.) Boiss.
quaking aspen	*Populus tremuloides* Michx.
Queensland kauri	*Agathis robusta* F.M. Bailey
radiata pine	*Pinus radiata* D. Don
red alder	*Alnus rubra* Bong.
red maple	*Acer rubrum* L.
red oak	*Quercus rubra* L.
red pine	*Pinus resinosa* Ait.
red spruce	*Picea rubens* Sarg.
rimu	*Dacrydium cupressinum* Sol. ex Lambert
rose	*Rosa*
rubber tree	*Hevea*
Sakhalin spruce	*Picea glehnii* (Schmidt) Mast.
sand pine	*Pinus clausa* (Chapm.) Vasey
scarlet oak	*Quercus coccinea* Michx.
Scots pine	*Pinus sylvestris* L.
senna	*Cassia*
sequoia	*Sequoiadendron*
Siberian larch	*Larix sibirica* Ledeb.
silver birch	*Betula verrucosa* Ehrh.
silver fir	*Abies alba* Mill.
silver-beech	*Nothofagus menziesii* (Hook. f.) Oerst.
silver maple	*Acer saccharinum* L.
silver wattle	*Acacia dealbata* Link
Sitka spruce	*Picea sitchensis* (Bong.) Carr.
slash pine	*Pinus elliottii* Engelm.
snowbrush	*Ceanothus velutinus* Dougl.
sour cherry	*Prunus cerasus* L.
sour orange	*Citrus aurantium* L.
southern blue gum	*Eucalyptus globulus* Labill. ssp. *bicostata* (Maid et al.) Kirkp.
southern-beech	*Nothofagus*
spike barkclothtree	*Brachystegia spiciformis* Benth.
spruce	*Picea*
stonefruits, apricot, cherry, peach, plum	*Prunus*
strawberry	*Fragaria*
subalpine fir	*Abies lasiocarpa* (Hook.) Nutt.
Sudan colanut	*Cola acuminata* (Pal.) Schott & Endl.
sugar maple	*Acer saccharum* Marsh.
sugarcane	*Saccharum officinarum* L.
sunbush	*Bossiaea*
sunbush	*Bossiaea laidlawiana* Tovey & Morris
swamp mahogany	*Eucalyptus robusta* Sm.
sweet orange	*Citrus sinensis* Osbeck
sweetcane	*Saccharum*
sycamore	*Platanus occidentalis* L.
sycamore	*Platanus*
tawa	*Beilschmiedia tawa* (Cunn.) Kirk
tawa	*Beilschmiedia*
tea	*Camellia*
teak	*Tectona*
terminalia	*Terminalia*
thuja	*Thuja*
tomato	*Lycopersicon*
toon	*Toona*
tung	*Aleurites*
tungoiltree	*Aleurites fordii* Hemsley
turkey oak	*Quercus cerris* L.
walnut	*Juglans*
wandoo	*Eucalyptus wandoo* Blakely
western hemlock	*Tsuga heterophylla* (Rafn.) Sarg.
western larch	*Larix occidentalis* Nutt.
western redcedar	*Thuja plicata* Donn ex D. Don
western white pine	*Pinus monticola* Dougl. ex D. Don
white fir	*Abies concolor* (Gord. & Glend.) Lindl. ex Hildebr.
white mulberry	*Morus alba* L.
white oak	*Quercus alba* L.
white spruce	*Picea glauca* (Moench) Voss
willow	*Salix*

Scientific Names — Common Names

SCIENTIFIC NAMES	COMMON NAMES
Abies	fir, true fir
A. alba Mill.	silver fir
A. balsamea (L.) Miller	balsam fir
A. concolor (Gord. & Glend.) Lindl. ex Hildebr.	white fir
A. grandis (Dougl. ex D. Don) Lindl.	grand fir
A. lasiocarpa (Hook.) Nutt.	subalpine fir
A. pindrow Royle	pindrow fir
Acacia	acacia
A. dealbata Link	silver wattle
A. decurrens (Wendl.) Willd.	green wattle
A. mangium Willd.	brown salwood
Acer	maple
A. pseudoplatanus	planetree
A. rubrum L.	red maple
A. saccharum Marsh.	sugar maple
A. saccharinum L.	silver maple
Actinidia	kiwifruit
Agathis	Agathis
A. australis Salisb.	Queensland kauri
A. robusta F. M. Bailey	kauri
Ailanthus	ailanthus
Albizzia	albizia
A. falcata Merr.	albizia
Aleurites	tung
A. fordii Hemsley	tungoiltree
Alnus	alder
A. rubra Bong.	red alder
Araucaria	araucaria
Arundinaria	cane
Asimina	pawpaw
Banksia	banksia
B. grandis Willd.	greatcone banksia
Beilschmiedia	tawa
B. tawa (Cunn.) Kirk	tawa
Beta	beet
Betula	birch
B. papyrifera Marsh.	paper birch
B. verrucosa Ehrh.	silver birch
Bossiaea	sunbush
B. laidlawiana Tovey & Morris	sunbush
Brachystegia	barkclothtree
B. spiciformis Benth.	spike barkclothtree
Callitris	cypress pine
Calocedrus	incense-cedar
C. decurrens (Torr.) Florin (*Libocedrus decurrens* Torr.)	incense-cedar
Camellia	tea
C. sinensis (L.) Ktze.	common tea
Carica	papaya
Carya	hickory
C. illinoiensis (Wangenh.) K. Koch	pecan
Cassia	senna
Castanea	chestnut
C. dentata (Marsh.) Borkh.	American chestnut
Casuarina	beefwood
Ceanothus	ceanothus
C. velutinus Dougl.	snowbrush
Cedrela	cedrela
Cedrus	cedar
C. deodora G. Don ex Loud.	deodar
Chamaecyparis	falsecypress
C. obtusa Endl.	hinoki
Cinchona	cinchona
Citrus	citrus
C. aurantifolia (Christmann in L.) Swingle	lime
C. aurantium L.	sour orange
C. sinensis Osbeck	sweet orange
Cocos	coconut
Coffea	coffee
Cola	colanut
C. acuminata (Pal.) Schott & Endl.	Sudan colanut
Corylus	hazelnut
Cryptomeria	cryptomeria
C. japonica (L.) D. Don	Japanese redcedar
Cupressus	cypress
C. lusitanica Mill.	Mexican cypress
Cytisus	broom
C. purgans (L.) Boiss.	provence broom
Dacrydium	dacrydium
D. cupressinum Sol. ex Lambert	rimu
Daucus carota L.	carrot
Diospyros	persimmon
Elaeis	oilpalm
E. guineensis	African oilpalm
Eucalyptus	eucalypt-gum
E. cypellocarpa L. Johnson	mountain grey gum
E. deglupta Blume	New Guinea gum
E. delegatensis R.T. Bak.	alpine ash
E. diversicolor F. Muell.	karri
E. dives Schau.	broad-leaved peppermint
E. fastigata Deane & Maid.	brown barrel
E. globulus Labill. ssp. *bicostata* (Maid et al.) Kirkp.	southern blue gum
E. grandis Hill ex Maid.	flooded gum
E. marginata Donn ex Smith	jarrah
E. obliqua L'Hérit.	messmate stringybark
E. radiata Sieb. ex DC.	narrow-leaved peppermint

SCIENTIFIC NAMES	COMMON NAMES
E. regnans F. Muell.	mountain ash
E. robusta Sm.	swamp mahogany
E. wandoo Blakely	wandoo
Fagus	beech
F. grandifolia Ehrh.	American beech
F. sylvatica L.	common beech
Ficus	fig
F. carica L.	common fig
Fragaria	strawberry
Fraxinus	ash
F. excelsior L.	common ash
Galeola	orchid
G. septentrionalis Reichb. f.	orchid
Gastrodia	orchid
G. elata Bl.	orchid
G. cunninghamii Hook. f.	orchid
Gliricidia	gliricidia
Gmelina	gmelina
G. arborea L.	gmelina
Gossypium	cotton
Grevillea	grevillea
Hevea	rubber tree
Humulus	hops
Hydnocarpus	chaulmoogratree
H. anthelminticus Pierre	common chaulmoogratree
Juglans	walnut
J. regia L.	Persian walnut
J. hindsii Jeps. ex Smith	northern California walnut
Khaya	khaya
Larix	larch
L. decidua Mill.	European larch
L. leptolepis (Sieb. & Zucc.) Gord.	Japanese larch
L. occidentalis Nutt.	western larch
L. sibirica Ledeb.	Siberian larch
Lavandula	lavender
Leucaena	leadtree
Libotrya	loquat
Litchi	litchi
Lycopersicon	tomato
Macadamia	macadamia
Malus	apple, pome fruit
Mangifera	mango
Manihot	cassava
Metasequoia	dawn redwood
Metrosideras	irontree
M. polymorpha (Gaug.) Rock	ohia
Monotropa	Indian pipe
M. hypopitys L.	Indian pipe
M. uniflora L.	Indian pipe
Morus	mulberry
M. alba L.	white mulberry
Musa	banana
Nothofagus	southern-beech
N. cunninghamii (Hook. f.) Oerst.	myrtle-beech
N. menziesii (Hook. f.) Oerst.	silver-beech
Olea	olive
Opuntia	cactus
O. ficus-indica Mill.	Indian fig
Paraserianthes	paraserianthes
P. falcataria (L.) I. Nielsen	paraserianthes
Parinarium	Parinarium
P. mobola F. Muell. ex Benth.	Muula
Passiflora	passion fruit, granadilla
Pastinaca	parsnip
Pelargonium	geranium
P. hortorum Bailey	fish pelargonium
Persea	avocado
Picea	spruce
P. abies (L.) Karst.	Norway spruce
P. engelmanni Parry ex Engelm.	Engelmann spruce
P. glauca (Moench) Voss	white spruce
P. glehnii (Schmidt) Mast.	Sakhalin spruce
P. mariana (Mill.) B.S.P.	black spruce
P. morinda Link	Morinda spruce
P. rubens Sarg.	red spruce
P. sitchensis (Bong.) Carr.	Sitka spruce
Pinus	pine
P. arizonica Engelm.	Arizona pine
P. banksiana Lamb.	jack pine
P. caribaea Morelet	Caribbean pine
P. caribaea var. *bahamensis* Barr. ex Golf.	Bahaman pine
P. caribaea var. *hondurensis* Barr. & Golf.	Honduran pine
P. clausa (Chapm.) Vasey	sand pine
P. contorta Dougl. ex Loud.	lodgepole pine
P. elliottii Engelm.	slash pine
P. kesiya Boyle ex Gordon (*P. insularis* Endl.)	Khasi pine
P. koraiensis Sieb. & Zucc.	Korean pine
P. monticola Dougl. ex D. Don	western white pine
P. nigra var. *maritima* (Ait.) Melville	Corsican pine
P. patula Schiede & Deppe	Mexican weeping pine
P. pinaster Ait.	Maritime pine
P. ponderosa Dougl. ex Laws.	ponderosa pine
P. ponderosa var. *arizonica* (Engelm.) Shaw	Arizona pine
P. radiata D. Don	radiata pine
P. resinosa Ait.	red pine
P. strobus L.	eastern white pine
P. sylvestris L.	Scots pine
P. taeda L.	loblolly pine
P. uncinata Mill. ex Mirb.	mountain pine

SCIENTIFIC NAMES	COMMON NAMES
Pistacia	pistachio
P. vers L.	common pistachio
Platanus	sycamore
P. occidentalis L.	sycamore
Populus	poplar
P. balsamifera L.	balsam poplar
P. grandidentata Michx.	bigtooth aspen
P. tremuloides Michx.	quaking aspen
Prunus	stonefruits, apricot cherry, peach, plum
P. amygdalus Batsch.	almond
P. armeniaca L.	apricot
P. cerasus L.	sour cherry
P. mahaleb L.	Mahaleb cherry
P. persica Sieb. & Zucc.	peach
Pseudotsuga	Douglas-fir
P. menziesii (Mirb.) Franco	Douglas-fir
Psidium	guava
Punica	pomegranite
P. granatum L.	common pomegranite
Pyrus	pear, pome fruit
Quercus	oak
Q. alba L.	white oak
Q. cerris L.	turkey oak
Q. coccinea Michx.	scarlet oak
Q. frainetto Ten.	Hungarian oak
Q. pubescens Willd.	downy oak
Q. robur L. (*Q. pendunculata* Ehrh).	English oak
Q. rubra L.	red oak
Q. suber L.	cork oak
Q. velutina Lamarck	black oak
Quinine	cinchona
Ribes	currant, gooseberry
R. nigrum L.	black currant
Robinia	locust
Rosa	rose
Rubus	blackberry, raspberry
R. loganobaccus L. Bailey	loganberry
Saccharum	sweetcane
S. officinarum L.	sugarcane
Salix	willow
S. matsudana Koidz.	Hankow willow
Sequoiadendron	sequoia
Solanum	nightshade
S. tuberosum L.	potato
Swietenia	mahogany
S. macrophylla King	Honduras mahogany
Tectona	teak
T. grandis L. f.	common teak
Terminalia	terminalia
Theobroma	chocolatetree
T. cacao L.	cacao
Theobroma	cocoa
Thuja	thuja
T. plicata Donn ex D. Don	western redcedar
Toona	toon
Tsuga	hemlock
T. canadensis (L.) Carr.	eastern hemlock
T. heterophylla (Rafn.) Sarg.	western hemlock
T. mertensiana (Bong.) Carr.	mountain hemlock
Ulmus	elm
Vitis	grape
Widdringtonia	cypress pine
W. whytei Rendle	mlanji cedar
Zizyphus	jujube

About the Authors

Charles G. Shaw III, better known as Terry, is Principal Research Plant Pathologist and Project Leader for the USDA Forest Service's research project on pest impact assessment technology. From 1977 to 1986, he was principal research plant pathologist at the Forest Service's Forestry Sciences Laboratory in Juneau, Alaska. He received a B.S. degree in forestry from Washington State University in 1970 and a Ph.D. in plant pathology from Oregon State University in 1974. His thesis dealt with Armillaria root disease in ponderosa pine. He continued working on that disease from 1974 to 1977 while serving as a research scientist with the Forest Research Institute in Rotorua, New Zealand, and as part of his research in Alaska. During 1982-1983 he had a 6-month assignment with the Pathology Branch of the British Forestry Commission, where he was able to establish a worldwide culture collection of various species of *Armillaria*. He currently is involved with root disease research in the Southwestern United States, and with efforts to model the dynamics, behavior, and impact of various forest pests, including Armillaria root disease. Address: USDA Forest Service, Rocky Mountain Forest and Range Experiment Station, 240 West Prospect, Fort Collins, CO 80526, USA.

Glen A. Kile is a Senior Principal Research Scientist and Program Leader, CSIRO Division of Forestry and Forest Products. A Ph.D. graduate from the University of Tasmania (1972), his research with CSIRO has been concerned with etiology and epidemiology of diseases in native forests — *Armillaria* and stem rot fungi in eucalypts, and *Chalara australis* in *Nothofagus* rainforest. He discovered *Armillaria luteobubalina* is a major pathogen in dry sclerophyll eucalypt forests, amenity tree plantings, and in horticulture in Australia, and has undertaken major taxonomic and population studies of *Armillaria*. He was Deputy Chairman and Chairman of the IUFRO Working Party on Root and Butt Rots of Forest Trees 1978-88. Address: CSIRO, Division of Forestry and Forest Products, Stowell Avenue, Battery Point, Tasmania 7004, Australia

James B. Anderson is a Professor of Botany at the University of Toronto, Erindale Campus. He obtained his B.A. in biology from the University of Rhode Island in 1975 and his Ph.D. in botany from the University of Vermont in 1980 shortly before he moved to his present appointment in Toronto. Dr. Anderson's research is concerned with the genetic control of mating and the process of speciation in higher fungi. His Ph.D. work was on sexual incompatibility and intersterility in *Armillaria*. Recently he has used molecular-genetic markers to examine phylogenetic relationships among species, and dispersal patterns in local environments. His current objective is to clone the mating-type genes of *Armillaria* and to compare their organization and expression among different strains and species of *Armillaria*. Address: University of Toronto, Department of Botany, Erindale College, Mississuaga, Ontario, L5L 1C6 Canada .

Harold H. Burdsall, Jr. is presently investigating the taxonomy and ecology of species of *Armillaria* and other forest fungi at the Center for Forest Mycology Research, USDA Forest Service, Madison. He received his B.A. degree in botany (1962) from Miami University and Ph.D. degree in mycology and plant pathology (1967) from Cornell University. He held a position with the Beltsville Forest Disease Laboratory (1967-71), then joined the Center for Forest Mycology Research in 1971. He became Project Leader of the Center in 1982. He has authored numerous papers on the taxonomy, nomenclature, and ecology of wood-rotting fungi. Current specific research interests concern species recognition in *Armillaria* using immunological and biochemical techniques. He is a past President of the Mycological Society of America. Address: USDA Forest Service, Forest Products Laboratory, One Gifford Pinchot Drive, Madison, WI 53705-2398, USA.

James W. Byler, Ph.D. University of California, Berkeley (1970), is currently Pathology Supervisor with the USDA Forest Service Northern Region at Missoula, Montana. His experience with Armillaria root disease

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Gregory M. Filip is an Associate Professor of Forestry at Oregon State University in Corvallis, Oregon. Before joining Oregon State University in 1990, Dr. Filip was a Research Plant Pathologist with the USDA Forest Service, Pacific Northwest Research Station, in La Grande, Oregon. He completed a Ph. D at Oregon State University in 1976 on chemical applications for control of Armillaria root disease of ponderosa pine. For the past 20 years he has been involved in research concerning the effects of silvicultural systems on biology and control of Armillaria root disease in the Pacific Northwest region of the USA. Address: Department of Forest Science, College of Forestry, Oregon State University, Corvallis, Oregon 97331, USA.

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Index